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ON THE MECHANISM OF ABSORPTION OF GRANULAR MATERIALS FROM THE PERITONEUM.

By W. G. MacCallum,

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As is well known there has been for a long time a desultory discussion as to the manner in which various materials are absorbed from the peritoneum and even in the case of solutions, it does not yet seem quite clear whether they are absorbed exclusively by the lymphatics or very largely by the veins. In the explanation of the absorption of granular materials we have an even more complicated process, which indeed illustrates always, one may say, the adaptation of the organism to a pathological condition, for one really cannot speak of an absorption of granular material from the peritoneum under normal conditions. Even the presence of red blood corpuscles, or leucocytes, or desquamated epithelial cells in the peritoneal cavity is in reality an abnormal condition and their removal must be effected by some means which form part of the reserve resources of the organism in the restoration of the normal conditions. It would seem *a priori* improbable that a special structure of the peritoneal walls should be arranged for the accommodation of granules of a certain size which might gain access to the peritoneum.

The question of the absorption of fluids has been discussed recently by Hamburger,[1] Cohnstein,[2] Adler and Meltzer,[3] Schnitzler and Ewald,[4] Starling and Tubby,[5] Mendel,[6] and others, the problem being mainly the determination of the relative amounts absorbed through the lymphatics and through the veins.

This problem is of course of the most fundamental importance, first of all in its bearing on the relation in function between the lymphatic system in general and the veins. As has been frequently stated, and very clearly defined by

[1] Centralblatt für Physiologie, 1895, ix, 484.
[2] Centralblatt für Physiologie, 1895, ix, 401.
[3] Journal of Experimental Medicine, Vol. 1, Jour. Physiol. 1897-8, xxii.
[4] Zeitschrift für Chirurgie, 1895. Wien klin. Rundschau, 1895, s. 273.
[5] Journal of Physiology, 1897-8, xxii.
[6] American Journal of Physiology, Vol. ii, 1898, p. xvi.

Dr. Sabin in a recent paper, the function of the lymphatic system is largely in the presentation of a channel in which the pressure is that of the veins near the heart, a pressure well known to be lower than that in the peripheral veins and into which the fluids of the tissues, in which the pressure is probably somewhere between that in the veins and that in the lymphatics, may drain. Now if it be found that nevertheless the chief absorption of fluid is carried out by the veins, we have the lymphatics occupying somewhat of a sinecure. The question is practically identical with that of the distribution of fluid injected into the subcutaneous tissue of a limb, in which the solution of the problem would probably be easier than in the peritoneum where it has been especially studied.

As to the mechanism of the entrance of the fluid into any of these channels by direct filtration, under the laws of osmosis and mechanical pressure, there is no great difficulty. It is not, however, so simple to explain the method by which insoluble granules gain entrance into the lymphatics. That they do so with great rapidity has long been known, although the various observers have held directly opposite opinions as to the mechanism of this absorption. v. Recklinghausen,[7] after demonstrating that such absorption did take place, arranged a rabbit's diaphragm on the stage of the microscope and covered it with milk. In his paper he states that he could clearly see the globules of the milk running in a vortex into holes in the serous lining of the diaphragm, leading down into the underlying lymphatics. These holes he was able to find afterwards in the same preparation and succeeded in outlining them with silver. With such large stomata the idea of the absorption of granules is a simple enough matter. No one, as far as I know, has ever succeeded in repeating this experiment although every one quotes him, and very many conclusions are based upon the observation. I have myself performed the experiment repeatedly—always with quite negative results—the milk runs into the grooves left between the tendinous fibers by the collapse of the lymphatics, but on washing off the surface all of it is washed off.

Two ideas have been in general vogue, as is well known, concerning the openings in the peritoneum. On the one hand the so-called stomata are generally thought of as openings of considerable size, surrounded more or less regularly by a radial arrangement of cells which are often somewhat differentiated from their neighbors and often have their nuclei situated in the end of the cell forming the boundary of the hole. On the other hand the stigmata with which the name of Arnold has been frequently associated are conceived of as much more minute openings which may occur along the line of union of adjacent epithelial cells, or preferably at the point of union of several of these cells. They are made evident by the application of silver nitrate, which leaves them as black dots or rings.

[7] Virchow, Archiv, 1865, Bd. 26.

The workers in Ludwig's laboratory did not succeed in confirming v. Recklinghausen's statements as to the presence of definite preformed stomata in the diaphragm of higher animals, although Schweigger-Seidel and Dogiel did see curious little pits lined with specialized cells in the membrane separating the peritoneum from the dorsal lymph-sac in the frog. Even here, later writers have found that these are only apparent openings.

It is interesting to note that from the perusal of the literature it is almost impossible to get any clear picture of the larger preformed stomata, which have been generally assumed in the text-books as the openings of the lymphatics. The most accurate description is that of the openings between the peritoneum and the dorsal lymph-sac in the frog but these have later been proven to be closed by cells. Ranvier[8] pictured certain "puits lymphatiques" which he at first thought to be openings but later he states that they are closed by mobile cells, related to the leucocytes. These were in the diaphragm of rabbits. No one has described accurately, or drawn the actual communicating channel between the peritoneum and the lymphatic, as one might expect to find it in a section and we are left with a very vague and uncertain conception of the nature of these openings.

The question of the rings and dots, the so-called stigmata, in the silver markings outlining the epithelial cells, has been widely discussed, some stating that they are merely irregular deposits of silver while others claim that they represent actual holes. A middle position is held by other writers who think that they may represent the openings produced by various things, such as the stretching of the serosa, or the perforation of the membrane by wandering leucocytes. Muscatello looks favorably upon this latter view but leans to the idea that if one could obtain an absolutely intact, normal serous surface there would be no "*stigmata.*"

It seems absurd that with all the work that has been done on the subject there should be any uncertainty about the existence of such stomata as v. Recklinghausen and many writers after him describe, and which are generally described in the text-books of histology to-day. They present such an easy explanation for the phenomena observed that they are eagerly seized upon as a useful structure by writers on the subject who make no further investigations for themselves. Of late, however, several authors have made the statement, that the lining of the diaphragm on the peritoneal surface is quite complete and contains no preformed stomata and that even the stigmata or irregularities in the silver markings are essentially artefacts. Kolossow[9] has made especially minute researches into the nature of the peritoneal epithelium and the connection of its cells, while Bizzozero and Salvioli[10] and Muscatello[11]

[8] Traite d'histologique.

[9] Archiv für mikroskop. Anat., 1893, Bd. 42.

[10] Archivio per le Scienze Medice, 1, 2 and 3.

[11] Virchow's Archiv, Bd. 142.

also describe the structure of the serosa and find no definite stomata. Other more recent writers however declare that stomata are present.

It is the aim of this paper to control these results which differ so widely in the interpretation of the anatomy of the peritoneal wall, and further to control the results of experiments made with the purpose of discovering the path taken by solid granules as they leave the peritoneum.

It is stated by Muscatello that absorption takes places almost exclusively through the diaphragm, as shown by the differences in the amount absorbed according to the position of the animal and that probably the parietal abdominal walls are very little concerned. His experiments as to this point are not numerous, however, and the tests which he applies to determine whether or not any of the material has been absorbed are not the most accurate. Nevertheless, even though the diaphragm is not the only part of the peritoneum engaged in absorption, it is generally agreed that it is perhaps the most actively absorbing portion. The determination of the exact paths of distribution of recognizable material placed at certain definite spots in the peritoneal cavity would probably afford considerable light upon this point, as was roughly demonstrated in the study of certain actinomycotic infections of the peritoneum.[12] In those rabbits in which the actinomyces produced a recognizable nodule wherever the individual organism or a group of organisms settled, intra-peritoneal injection of a suspension resulted invariably in such a dense production of the minute white nodules over the diaphragmatic tendon that its surface was almost entirely covered. In all cases the omentum was densely studded with the nodules and contracted into a firm mass. There were generally abundant nodules in the pelvis but the parietal abdominal walls, mesentery and intestines were usually almost free or the seat of scattered lesions only. On the other hand the injection of carmine suspension into the peritoneum which results in an injection of the diaphragmatic lymphatics and the filling up of the anterior mediastinal lymph glands, also produces a diffuse pink staining of the parietal abdominal walls which on microscopical examination is found to be due to the presence of finest granules of carmine in the tissue, generally inclosed in leucocytes.

Very little stress is laid by all these writers upon the enormously active phagocytosis, which may be observed in the peritoneal cavity a short time after the introduction of granular foreign material.

This may be studied easily enough by examining the peritoneal fluid of dogs at various intervals after the introduction of a suspension of granules. There are quite marked individual differences in different animals in the rapidity with which phagocytosis occurs. Generally it has been found that after fifteen to twenty minutes very few leucocytes are to be found in the peritoneal fluid. Some of these are actually laden with pigment while others have applied themselves to masses of pigment. After the lapse of thirty minutes the activity of the endothelial and epithelial cells becomes more marked and in two dogs forty-five minutes after the introduction of the material the leucocytes, which usually are not excessively abundant at thirty minutes, had become extremely numerous and were abundantly laden with pigment. It has been found, however, that the entrance of bodies, such as extravasated red corpuscles, into the lymphatics may take place before any great exudation of leucocytes has appeared—for example in one case in which a vessel was wounded during the injection, large enough to fill the peritoneum with blood, the dog was killed twenty minutes after the injection—hardly any phagocytic leucocytes could be found but the lymphatics and the mediastinal lymph glands were full of blood.

Whether the diaphragm be the most important absorbing agent or not, it is a situation in which the study of the mechanism of absorption into the lymphatics has long been carried out and it is again with lymphatic absorption as typified here that this study is almost exclusively occupied.

In order to comprehend the mechanism of absorption it is necessary first to study in its detail the anatomy of the tissues concerned, and more especially of the tissues, if any, which separate the lumen of the lymphatic channel from the cavity of the peritoneum. It will be readily seen that the crucial point is the passage of the granules through this tissue, for once within the lymphatic the transposition of the material is an easy matter, all the mechanical conditions being entirely favorable to the progression along certain lines.

The pleural surface of the diaphragm may be readily distinguished from the peritoneal by various anatomical peculiarities, a few of which will interest us. It is found that the connective tissue bundles which show through the transparent superficial tissue lie in general parallel with one another. Large blood-vessels and nerves course through this tissue and the lymphatic trunks can frequently be seen as clear canals, branching about over the surface. When injected they are found to have a characteristic arrangement which, however, need not be described here except in its merest outlines. There are lateral semicircular canals coursing along with the lateral veins near the line where the musculature passes over into the tendinous center. From these efferent trunks pass forward to the anterior mediastinum, where they accompany the mammary veins, while others run backward to the point where the vena cava, œsophagus, and aorta penetrate the diaphragm, and accompanying these structures, pass through also to empty into the abdominal lymphatic trunks.

Extending in all directions from these main trunks on the pleural surface are branching channels with the characteristic segmentation and provided with the curious long filmy valves at each segment, which flutter like flags in the stream and have a line of closure a long way from the actual

[12] MacCallum, Life History of Actinomyces Asteroides, Centralb. f. Bakt. u. Paras., 1902, xxxi.

edge. These channels anastomose abundantly and form a network over the whole surface. Passing through the musculature of the diaphragm, from this network, are short trunks which communicate with the layer of lymphatics lying on the peritoneal side. The tissues here are rather differently arranged—the muscle bundles run for the most part parallel with one another and radially, being separated by connective tissue in which lie the parallel and likewise radially placed lymphatic canals which receive the communicating channels from the pleural side. These canals are somewhat beaded or bulged along their course, but it could not be determined that they are provided with valves. They project a little above the muscle into the overlying connective tissue on the peritoneal side. The bundles of this connective tissue layer are in general parallel with one another but diverge here and there to leave lozenge-shaped spaces, which are readily visible to the naked eye in the dog and in man often measure 2-3 mm. in length and 1 mm. in width. The connective tissue bundles and therefore also these spaces run in a direction transversely or obliquely across the radially arranged muscle fibers.

Now the parallel lymphatic canals are most abundantly connected by anastomosing channels which run obliquely or transversely across the intervening muscle bundles and lie generally nearer the peritoneum than the radial trunks themselves. Often some of these running obliquely, join in the middle between two of the radial trunks to form a sort of secondary radial trunk which lies upon the surface of these muscle bundles. Others arch up toward the peritoneum in their course from one radial trunk to another. These are sometimes relatively simple fusiform tubes, while at other times they branch and interanastomose with one another in a complicated way, often forming secondary connections with other parts of the trunks by means of very narrow canals. In general all of these anastomosing channels open into the radial trunks by exceedingly wide and numerous openings, so that there is the freest possible intercommunication. In their arching course they come to lie in the spaces between the connective tissue fibers of the layer overlying the muscle, that is in those lozenge-shaped spaces which are described above, and here they are separated from the peritoneal cavity by an extraordinarily thin layer of tissue only. This somewhat complicated arrangement is perhaps best described by the drawing which shows the extremely rich network of vessels. (Fig. 1.)

It will be seen that these form the most favorable situations for the entrance of materials from the peritoneum and undoubtedly they are especially adapted to that purpose. These sac-like channels are easily seen with the naked eye, even without being injected or distended. They appear as small diamond-shaped or fusiform, clear areas, generally a little sunken below the surrounding tissue, but when distended they are elevated above the surroundings as tiny vesicles. In the diaphragm of man and the dog the covering of transparent tissue is easily seen. In the ox's diaphragm, however, they look like mere holes or depressions on the surface and it is only by picking up with a forceps out of the depth of the depression, the membranous roof, that we see the real nature of the structure. In the rabbit they are elongated and radially placed—especially developed in the centrum tendineum, where they lie between the radial fibers. In dogs and man, however, they seem more abundant over the musculature of the diaphragm.

Now when granular material is injected into the peritoneal cavity we find on examination of the diaphragm after a time that these superficial anastomoses or blind sacs, or, as we for brevity call them—lacunæ—are injected with the granular substance and from them we can trace the material into the anastomosing trunks of the pleural network, into the efferent trunks, and then very readily into the mediastinal lymph glands. It is interesting to note that even when actual entrance of the pigment into the lacunæ has not occurred it is deposited almost exclusively upon their roofs, possibly because in their collapsed condition they lie somewhat below the surrounding level. The roofs of these lacunae must, therefore, be of especial interest to us in determining the mechanism of the absorption.

It is found that while they lie between the connective tissue bundles and are thus situated obliquely or transversely over the muscle, they are themselves traversed by other finer fibrils which cross them obliquely or transversely and which consist no doubt, merely of diverging superficial fibers of the main layer. When, therefore, one looks down upon one of the lacunæ in its distended condition one sees these fibrils cross its bulging surface like ropes across a full sail. Elastic fibers are present in numbers, often forming definite layers throughout the superficial connective tissue of the diaphragm, and they too, often stretch across the roofs of the lacunæ.

The further structure of these tissues can be best made out in thin paraffin sections of tissue, fixed in Zenker's fluid, cut vertically and in sections parallel with the surface of the diaphragms, which have been treated with nitrate of silver or by the method of Kolossow.

The lymphatics themselves can be beautifully demonstrated by an injection of a 0.5% solution of silver nitrate, followed by agar which in cooling keeps the channels widely distended and makes it possible to view the smoothly stretched lining. (Fig. 1.) By this method it is found that the endothelial lining is an exquisitely complete one. Similar methods employed in demonstrating the continuity of the endothelial lining of the lymphatics of the skin were described in a previous paper but if possible the accuracy of the adjustment of the cells to one another is shown here even more beautifully than was the case in those channels. It is generally found that the lining endothelial cells are rather elongated in the radial trunks—often their outlines are zigzag and irregular. In the wall of the lacunæ nearest

the radial channels this irregularity of outline of the cells is very marked, while on the bulging roof they appear as if drawn out and stretched into more regular outlines. These cells of the roof are perhaps rather smaller than the others —they are long and narrow, their long diameter sometimes lying parallel with that of the channel, sometimes across it. Over the whole surface of the distended lacunæ, however, they are adjusted to one another with the utmost delicacy and accuracy—there being in the many lacunæ examined no signs of a preformed opening. Even the rings and dots of the silver deposit—the stigmata—are almost entirely lacking, the intercellular lines being extremely delicate and fine.

The question of those curious rings and dots which are produced by treating the endothelial and epithelial cell layers with silver is one which is not yet satisfactorily answered. Certainly they can hardly be dismissed as entirely the result of accidental irregularities in the action of the silver, although this may account for the grosser of these deposits in cases in which the tissue was not previously carefully washed, or in which too strong a solution was used and allowed to remain too long in contact with the tissue or insufficiently removed by washing with distilled water. In these cases there appear black granules and masses which are apparently due to the presence of albuminous fluids upon the surface or to the gross precipitation of the silver from the solution on long contact with the tissue. If all these precautions are observed, however, and a solution of silver nitrate used which is not more concentrated than 0.5 per cent, such gross precipitates are avoided. Further, as has been repeatedly pointed out, stretching, drying, inflammation and a great variety of other processes injurious to the tissue favor the production of these appearances. So, for example, in serous surfaces in which the rather violent stretching action of artificial respiration has continued for a long time, it is usual to find the inter-epithelial lines much widened, especially over the lacunæ.

Even with the exercise of every precaution, however, there appear some of these markings—in the epithelial layer they take the form of solid widenings of the intercellular line or of rings intercalated in these lines. No doubt these may be explained as due to a partial separation of the cells—an enlargement of the intercellular space which, as Kolossow has shown, normally exists and a separation of the intercellular bridges which cross this space. Whether this will explain all of them is uncertain, but there seems no doubt that the epithelial cells can easily become separated and then there are conditions produced which favor the greater deposit of the silver between them.

In the endothelium of the lymphatics the same minute rings appear in small numbers along the intercellular lines. Now Kolossow insists that the type of union of the endothelial cells is quite similar to that of the epithelial and we have no reason to doubt that this silver deposit would react in the same way there. There is, however, another sort of ring intercalated in the intercellular line which may best be explained in another way. These are not like the minute darkly outlined rings just described, but larger and bounded by a more or less irregular outline. Their central portion is granular, resembling exactly the bodies of the endothelial cells—indeed these rings look like very small cells or parts of cells intercalated in the endothelial line and it seems most probable that they are actually parts of endothelial cells nipped off as it were by the gradual narrowing of the neck of one of the numerous processes of the original cells, as shown in Fig. 2, in which from A to E one may trace the progress of formation of such rings. They no doubt form an integral portion of the wall of the lymphatic—sometimes they may actually appear to lie in the body of one of the larger cells and connected with its margin only by the line along which the edges of the large cells have come together so as to completely isolate this small mass. (Fig. 2 E.)

This forms one structure of the roof of the lacunæ. Another important one is the layer of peritoneal epithelial cells, which have given rise for a long time to the most vigorous discussion. Their nature has been investigated by a great variety of methods, with a very remarkable variety of results. They have been described as flattened cells arranged edge to edge—the method of approximation of the edges remaining a little obscure. It has long been the opinion of various authors that the cells are fixed to one another by a sort of cement which on being treated with silver nitrate forms with that reagent a silver albuminate which blackens on exposure to light. These authors have

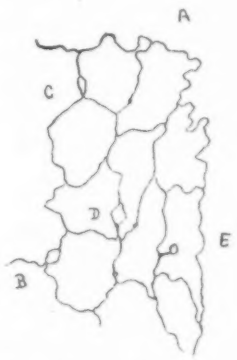

FIG. 2.—Endothelium of a lymphatic lacuna showing the process of separation of small portions of the cell body from the elongated processes.

described them as polygonal cells, ordinarily homogeneously arranged, but sometimes arranged about an opening in which case the nuclei of the cells approach the opening. In other instances certain groups of cells are smaller and more densely granular than the remainder—these are thought of as germinating cells and so described by Klein.[13]

The only advance in knowledge of the subject in recent years has been made by Kolossow,[14] who carried out a detailed study of these cells by the aid of a method in which osmic acid was used as the fixative agent, followed by a tannic and pyrogallic acid solution, the action of which is analogous to that of the photographic developers. By this means he showed that a much more complicated structure than had been previously supposed existed in the epithelial cells. He found that the cell body containing the nucleus was of granular protoplasm and relatively small,

[13] Atlas of Histology.

[14] Archiv für mikr. Anat., 1892, Bd. 42.

being quite widely separated from the adjacent cells and connected with them only by protoplasmic processes or bridges. Over these gaps, however, the perfectly hyaline, thin, flat, superficial layer of the cells extended like the widely projecting eaves of a flat-topped house, to meet or almost meet a similar projecting layer from the next cell. These hyaline plates, too, are connected by intercellular bridges and are covered over on their free surfaces with long thin cilia. When such cells are looked at from above, the fine line between the hyaline plates is scarcely visible, but the slightly irregular margin of the underlying granular cell with its protoplasmic bridges, shows through and one sees between the cells a beaded line of clear spaces which are the spaces between the granular intercellular bridges, showing through the hyaline plate.

Everything in this description which has been accepted also by Muscatello has been confirmed in our studies—the cilia are thickly set on the surface and quite conspicuous in sections. It may be questioned, however, whether there is not some shrinkage in the cells so treated to produce the wide gaps between their protoplasmic portions. Indeed in several instances the picture produced has been rather that of a series of stellate cells connected only by the long processes which stretch out from the protoplasm, tightly contracted about the nucleus. The gaps thus left between the cells are very large indeed.

With the same method it is sometimes observed that the cells contract away from one another and assume in sections an almost spherical form. It has often been observed, however, in preparations of inflamed serous surfaces fixed in Zenker's fluid that the cells do remain in contact superficially, while their cell bodies are separated and we have even found these spaces occupied by leucocytes.

Both Kolossow and Muscatello found by this method, which is more refined than the silver method in that irregular precipitates are avoided, that the peritoneal epithelial lining is a complete one. They fail entirely to find any apertures with special arrangement of epithelial cells about their margins, such as have been described by several authors, and they see nothing of the groups of germinal cells spoken of by Klein and others. Briefly it may be stated that our results agree entirely with those of Kolossow and Muscatello, for search through a great number of preparations has failed to reveal anything which could be considered a preformed hole in the peritoneal lining. An especially careful scrutiny of the roofs of the lacunæ in the dog's diaphragm was made but nowhere in the hundreds of roofs examined could any such aperture be found—the frequently described radial arrangement of cells about a central hole being entirely wanting. The partial separation of the cells from the contraction of their protoplasm, as described above, has, however, its significance, as will appear later.

What then, we must ask, is the nature of the tissue which lies between the peritoneal epithelium and the endothelium of the lymphatics. Bizzozero and Salvioli,[15] Vincenzi,[16] and Muscatello [17] have directed their attention especially to this point and agree in describing a homogeneous basement membrane which lies directly beneath the epithelium. Bizzozero and Salvioli determined the presence of this by maceration of the tissue in Muller's fluid and alcohol, after which they were able to tear off the membrane in small strips. This they found to be imperforate and homogeneous everywhere except in those areas in which it formed part of the roof of the lymphatics in the diaphragm and there it was pierced with numerous fenestrations. Vincenzi found similar fenestrations in the membrane lining the tunica vaginalis testis. Muscatello adopted a similar method of investigation and studied the membrane in many situations in the peritoneum. He found that it was easy to strip off quite large pieces from the intestine and several other organs, while from some others in which the tissues were denser, it became far more difficult. He was able to see niches and depressions in the surface of this membrane in which the epithelial cells lay. Only over the lymphatics of the diaphragm could he find any fenestrations and from this he draws his conclusion that it is only in the diaphragm that the absorption of granular material occurs.

Maceration as practiced by these authors has given us no very satisfactory results, but some light upon this point has been obtained by the study of flat and vertical sections treated in various ways, the results of which may be detailed briefly. The fibrous tissue which makes up the bulk of that layer in which the lymphatics lie embedded gives off, as described above, divergent bundles which run obliquely across the lymphatics and thus come to lie in the substance of their roofs. Sometimes these strands are so abundant that the cross section of the roof of such a lacuna looks like a string of beads and from the projection of the strands into the lumen of the lacuna its cavity becomes very irregular in form. This fibrous tissue is extraordinarily rich in elastic fibres, which lie in more or less definite layers and accompany the fibrous strands into the roof of each lacuna. From the coarser elastic fibres accompanying these strands, fine branches are given off which run in all directions over the roof of the lacuna so that it becomes very richly supplied with elastic tissue. If now we examine a flat section stained with Van Gieson's or Mallory's stain for connective tissue, or still better one treated with a combination of silver nitrate with Kolossow's osmic-tannic acid stain, all of which leave the elastic fibres very inconspicuous, we can make out in the roofs of the lacunæ between the epithelium and endothelium a lattice work of fibres which has a general resemblance to a fenestrated membrane. (Fig. 3.) Close examination shows, however, that it really consists of anastomosing bundles of fibrils which form more or less rounded meshes. These fibrils stain rather pale blue with Mallory's method—

[15] Bizzozero and Salvioli, Archivio per le Scienze Medice, 1, 2, 3.
[16] Vincenzi, ibid.
[17] Muscatello, loc. cit.

pale red with Van Gieson's and stand out sharply as greenish grey on the application of the other method described. In vertical sections the lattice work appears as a rather thick band directly underlying the epithelium in which sometimes all the cords of fibrils are cut across so that we have the appearance of a beaded line. (Fig. 6.) In other cases one of the strands may be cut longitudinally and the others obliquely, producing variations in appearance. The coarse fibrous strands interrupt this line or they may lie beneath it, holding the endothelium of the lymphatics away from it. They are often invested by its prolongations. (Fig. 6.) At the margin of the lacunæ, the lattice work thins away and runs on beneath the epithelium and over the most superficial layer of elastic fibrils. Everywhere, aside from the lacunæ, the epithelium is bounded below by the extremely delicate basement membrane which appears in section as the finest line, even when viewed with the oil immersion objective. Whether the lattice fibers in the roof of the lacunæ are directly continuous with this I cannot definitely determine, but one receives the impression that this is the case. It seems inconceivable that even with the most careful maceration this extraordinarily delicate basement membrane could be torn off in pieces of any size in an entirely isolated condition. Our most successful isolation preparations included always a good deal of the underlying tissue.

Elastic fibrils and scattered connective tissue cells make up the remainder of the tissue which separates the epithelium from the endothelium of the lymphatic—a tissue which is thus seen to be far from compact and which on section shows abundant wide crevices and spaces the significance of which will also appear later.

The endothelium in these sections passes as an extremely delicate line over the whole inner surface, bulged here and there by a relatively thick nucleus and sinking into the spaces in the lattice work or held up by the coarse fibrous strands. According to the degree of distension of the lacuna this irregularity is greater or less. When the lacuna is widely distended the endothelium is drawn out smoothly over the tissues of the roof and all irregularities are obliterated. Study of very many sections shows no evidence of break in the continuity of this layer in spite of its irregularity.

Further attempts were made to be sure that the roofs of the lacunæ are indeed without definite apertures. As stated above they never could be seen either in the peritoneal epithelium nor in the lymphatic endothelium, but it seemed that an irrefragable proof might be obtained if we could distend these lacunæ with a colored injection mass. Of course it can be conceived that a canal opening very obliquely into the lacunæ might be shut valve-like by the distension of the lacunæ, but it seems *a priori* extremely improbable that such an arrangement could exist, especially since the roof of the lacuna is so extraordinarily thin,—furthermore such a canal should be discoverable on section. At any rate bearing this in mind most numerous injections were made, generally under the stereoscopic microscope in the fresh diaphragm—it is easy enough to insert the needle into the superficial connective tissue on the peritoneal surface and inject two or three or more of the parallel trunks with all their anastomoses and blind appendages. Usually such an injection takes the form of a straight band running from the central tendon radially toward the periphery of the diaphragm, over which the obliquely placed lacunæ bulge up and produce a corrugated appearance on the surface. They may be distended to their greatest capacity, putting the fibrils which traverse them on the stretch and bulging between them as thin walled sacs. With the removal of the pressure they collapse and may be redistended over and over again. With all this, leakage does not necessarily occur—it is easy to inject the whole breadth of the diaphragm without the least leakage and even to gently distend the lacunæ. When the surface is exposed to the air until the moist glisten is slightly lessened it is especially evident that there is no leakage. In such a distended system one may with a camel's hair pencil brush the colored liquid out of one lacuna back into the trunk and into another lacuna, when with the relaxation of the pressure it returns to refill the first lacuna. With injections of air one can distend these lacunæ into shining bubbles which remain distended a long time. Certainly one receives the impression that they are sacs with complete walls and without any such holes as are described in their walls large enough for two or three red corpuscles to go through abreast. This is not the whole truth however, for by slightly raising the pressure one *can* make the fluid appear on the surface—it oozes through slowly, appearing like tiny dew-drops. If we use a solution, the fluid which comes through is colored too, but if we use a suspension such as carmine or milk, the fluid which filters through is clear, the suspended particles being left behind. This seems to be merely a process of filtration under pressure through a thin membrane and does not interfere with the idea of the completeness of the membrane.

If now we increase the pressure still more we may be able to observe suddenly a whirl of pigment granules bubbling up from the lacuna through the roof at some point and flowing over the surface. This is explosive in its appearance and it has seemed each time it was observed to be due to the rupture of the roof of the lacuna by the relatively high pressure employed. It is hard to conceive that these things should be possible if there were wide open communications between the lacunæ and the peritoneal cavity. Indeed it seems almost inevitable that were there any free openings at all there would appear droplets of milk, as such, on the surface or of the india ink, as such, instead of which the surface remains for a long time without any transudate, after which with continuance of the pressure there is a general oozing of clear fluid.

To resume then, we have the peritoneal cavity lined by a complete layer of peculiar epithelial cells which lie on a basement membrane uniformly thin except where it overlies

the lymphatic lacunæ in which position it is represented by a lattice work of fibrils separating the epithelium from the surface of the lymphatic. Approaching the peritoneum at these points are the oval sacs or lacunæ which are the absorbing terminals of the diaphragmatic lymphatics and which while possessed of a complete lining of endothelium are separated from the peritoneal cavity only by the loosely woven connective tissue and the peritoneal epithelium.

We have therefore to deal with at least three elements in the absorption of material from the peritoneum, no matter whether such materials pass directly through the roofs of the lacunæ or first through the peritoneal epithelium at a distance from them and then by a circuitous route through crevices in the tissue to the walls of the lymphatic channel. These three elements which can afford a certain obstruction to the progress of the absorbed material are then the peritoneal epithelial layer, the basement membrane and the lining endothelium of the lymphatic.

Now in the preceding paper, above referred to, it was shown that the walls of the lymphatics in several situations at least are composed of a complete layer of endothelial cells without preformed openings and when we come to explain the entrance of solid materials into such channels we are met by difficulties. The simplest and readiest explanation which was offered in that paper is that phagocytic cells possessed of automatic motility may carry the granules in, or the vital phagocytic activity of the endothelial lining cells may be capable of effecting an entrance for such granules. To explain the entrance of free granules it seems that we must have some propelling force and then we must have either open holes in the walls or the connection of the endothelial cells must be so lax that the force is sufficient to drive granules between them into the lumen. This last idea is the one adopted by Muscatello although it is certainly at first sight not a very satisfying one. It is the idea expressed also by Fleiner[18] in his elaborate researches on the absorption of pigment from the alveoli of the lung.

Mention has already been made of the extraordinary activity of the leucocytes as well as the fixed cells in taking up any foreign materials. If one inject a suspension of carmine into the peritoneum of a rabbit and after a few hours examine the diaphragm the lacunæ will be found sprinkled over with leucocytes and their lumina largely filled with them. Indeed if one dissolve out the carmine,—the acetic acid concerned in fixation with Zenker's fluid is sufficient to effect this—and then stain the diaphragm in toto in a nuclear stain, it will be found that the lacunæ appear densely injected with blue which on closer inspection is seen to be due to the leucocytes which crowd its lumen. So, too, we find the pleural network full of them and the lymph sinuses of the mediastinal lymph glands are practically packed with phagocytic leucocytes. So, too, in the dog—forty-five minutes after the injection of a large quantity of diluted india ink into the peritoneum the fluid in the peritoneum is found to be practically a thick suspension of amœboid leucocytes, a great many of which are laden with pigment granules. The lacunæ in the diaphragm are found to contain a great quantity of pigment, a large part of which is contained in leucocytes. Leucocytes generally laden with pigment swarm about over the surface of the lacunæ and such laden leucocytes can actually be seen in large numbers in sections *making their way with their load of pigment through the roof of the lacuna.* (Fig. 4.) These are, as shown in the drawing, greatly distorted and drawn out, their protoplasm and nucleus stretched out into a thread in the advance through the tissue and drawing behind usually a larger bulbous portion carrying the pigment. Often one can see four or five going through different points in the roof of one lacuna. That they are entering and not going out seems the only reasonable conclusion from the general direction of the stream, even if one cannot draw any absolute conclusions from the appearance of the leucocyte in transit.

The epithelial cells and especially those over the lacunæ are frequently phagocytic and become loaded with pigment. Similarly the connective tissue cells underlying them may often show a pigment content, especially when they lie just at the edges or on the roof of the lacuna. Finally the endothelial cells in the lymphatic become swollen and loaded with pigment.

From all this it may with reason be concluded that phagocytosis at least plays a very important part in the transportation of pigment from the peritoneal cavity to the cavity of the lymphatics, for not only have we leucocytes wandering into the lymphatics and carrying pigment but the epithelial and endothelial cells themselves take up the pigment and may perhaps be able to transfer it, one to the other. Leucocytes laden with pigment may also be seen wandering in the crevices of the tissue outside the lymphatic but doubtless these later enter the lymphatics also. We cannot of course tell when we find a leucocyte laden with pigment in the lymph gland that that leucocyte has brought the pigment from the peritoneum—it may have merely taken up free pigment brought to it in the lymph gland, but when we see leucocytes actually penetrating the barrier between the peritoneum and the lymphatic with its load of pigment there seems to be but little doubt of its significance.

The importance of the respiratory movement of the diaphragm in the aid of absorption from the peritoneum has always been emphasized and indeed it is not to be underrated, as will be seen later. The mechanism by which it produces a pumping action is simple enough—in inspiration when the diaphragm is relatively flat and contracted the spaces between its constituent muscles and connective tissue fibres become compressed and conversely in expiration, when it arches up into the thorax these elements separate and the intervening spaces are widened. It is very easy to see

[18] Fleiner, Virchow's Archiv, 1888, Bd. 112.

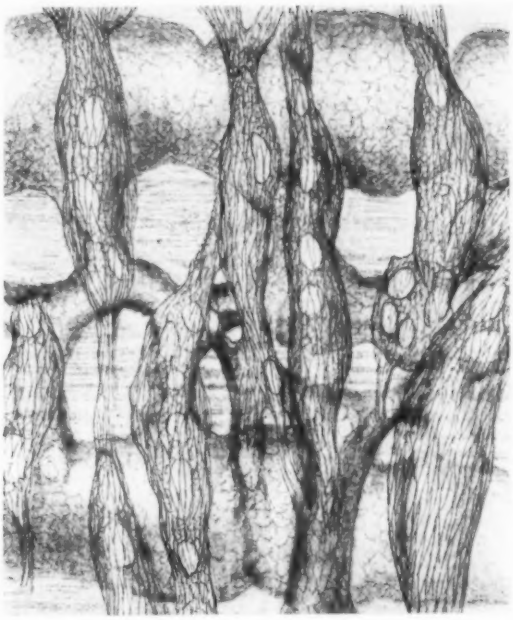

FIG. 1.—Diaphragm of dog in which the lymphatics have been injected with silver nitrate. The lacunæ are intended to appear as arching anastomoses between the radial trunks which lie at a lower level and to communicate with them freely by wide openings.

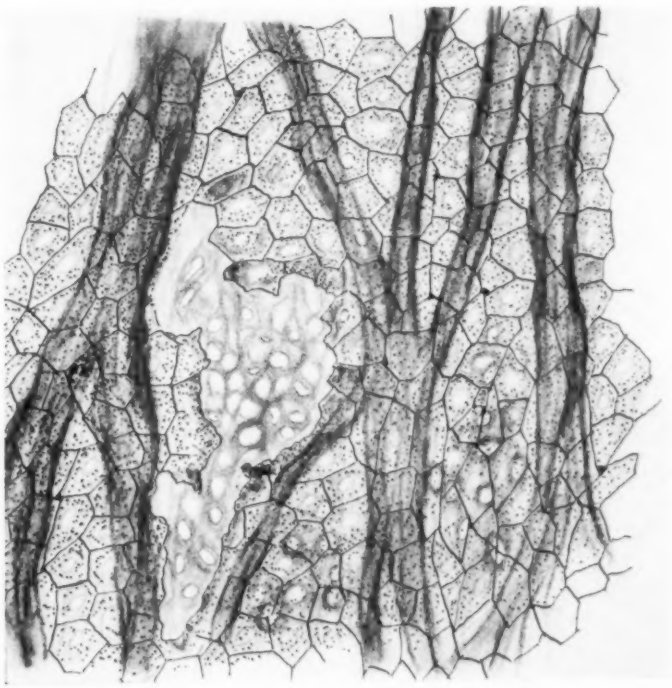

FIG. 3.—Superficial layer of diaphragm stained with silver nitrate, &c., showing the lattice work of fibres overlying the lacunæ.

therefore that the lymphatic channels which lie in these spaces are also alternately distended and compressed—in other words there is an actual pumping action carried on incessantly by the respiratory movements. In order to determine whether or not this is necessary to absorption in the living dog the elimination of respiratory movements were brought about by the wide opening of the chest. The lungs were drawn out as far as possible and active artificial respiration kept up, so active that no impulses came from the nervous system to the diaphragm to produce its contraction. When the dog was then tipped up by the elevation of the hind legs the diaphragm bulged slightly into the thoracic cavity and remained quite quiet. Carmine in suspension was then injected into the peritoneal cavity and the dog kept alive for two hours. At the autopsy it was always found that in spite of the absence of respiratory movements there was a fairly abundant injection of the lymphatic lacunæ and enough carmine reached the anterior mediastinal glands to color them bright red.

There seems no doubt therefore that the vital activity of the cells—phagocytosis—is capable of producing by itself an injection of the lymphatics and is of extreme importance in the absorption of granular material.

That it is not the only factor, however, is shown by the presence of free pigment granules within the lacunæ and in order to determine their mode of entrance it was decided to eliminate one by one the possible adjuvant factors.

The dog was killed and partly eviscerated and then hung up with artificial respiration so that the granular material might be poured upon the concave peritoneal side of the diaphragm which moved up and down with the expansion and contraction of the lungs. This eliminated in large part the agency of the leucocytes, since the peritoneal cavity was washed out, and in other experiments this factor was more absolutely excluded by washing out the blood-vessels with salt solution. On continuing the artificial respiration for two or three hours one generally succeeded in producing a very definite injection of the lymphatic lacunæ, the pleural network and of the mediastinal lymph glands. If before beginning the respiratory movements one portion of the diaphragmatic surface were brushed with a camel's hair pencil, it was found afterward that that area was much more deeply injected than the remainder. Evidently here one brushed off the roofs of the lacunæ and the pumping action of the diaphragm readily filled such violently opened channels. The possibility therefore suggests itself that even in the other case the absorption might be due to slight traumatisms injuring the epithelium of the peritoneal surface, and other experiments were instituted in which in the removal of the abdominal viscera extreme care and gentleness were observed, the diaphragm was not once approached and the liver left in situ. In this case, however, the injection of the lacunæ was quite as extensive and quite as general.

With the respiratory movement therefore it is possible to produce an injection of the lacunæ without the aid of the phagocytic leucocytes.

It may be objected, however, that the active phagocytosis displayed by the endothelial and epithelial cells may have aided greatly in this transmission of pigment. In order to exclude this, various reagents were applied to the surface of the diaphragm before the granular material was poured on with the aim of either paralyzing these cells or killing them outright. Solutions of quinine for example were used. Water heated to 65° C. was allowed to stand in the diaphragm for three to five minutes. Even a 10% solution of formalin was poured on the diaphragm and allowed to stand for two, five, and twenty minutes, respectively. In another instance silver nitrate was poured over the surface of the diaphragm. After this the granular material was poured on and artificial respiration carried on. In all of these cases a small amount of absorption occurred—certainly as might be expected after the use of such violent coagulating agents, the absorption was not as free as in the fresh diaphragm but some of the granules did gain access to the lymphatics and in some cases, e. g., after the formalin the mediastinal glands became deeply injected. Similarly a dog was killed and allowed to lie in the ice-box for four days until all phagocytic activity on the part of the peritoneal epithelium had probably been destroyed. In spite of extreme rigor mortis it was found that artificial respiration produced, when the dog was eviscerated and carmine poured on the diaphragm, some absorption of the pigment into the lacunæ. There was evidently so great a coagulation of the tissues that the lymphatic trunks and lymph glands in the anterior mediastinum did not become injected.

By this means we have eliminated everything which could bring about the absorption except the mechanical agency of the respiratory pumping movement. True it is, too, that in so doing we have lessened the amount of absorption to a small fraction of what it was when the living cells were aiding by their phagocytic activity but nevertheless there is left the fact that the absorption is not exclusively dependent on phagocytosis.

In order to determine the paths of the absorption all these diaphragms were fixed and numerous sections cut from various portions of each.

Practically two histological pictures only were obtained from all these various experiments—either phagocytosis played a part or it did not. In the first case the appearances have already been described and pictured—in the second one found the cells which go to make up the tissue practically passive and the path of the entering pigment can only be judged by the study of its distribution in the sections. Since the principle is identical in all of these cases in which the vital activity of the cells is excluded, it may be well for the sake of brevity to describe the results of one experiment in which the distribution is best seen. Such an experiment is that in which diluted india ink is poured upon the concave surface of the diaphragm of a

freshly killed dog and artificial respiration carried on for two or three hours. In such a case phagocytosis, if it occurs at all, is negligeable—no cell enclosing pigment has been found in any of the sections, probably largely because the cells still surviving are not kept warm and their amœboid motion ceases. The entrance of the ink in such a case therefore occurs just as it does in those cases in which the tissue cells are actually killed.

It is found that the epithelial cells are slightly contracted and rounded off so as to separate a little from one another. This is not invariable but is very common—sometimes it produces such a convexity of the free surface, of each cell that the cilia become separated and radially arranged.

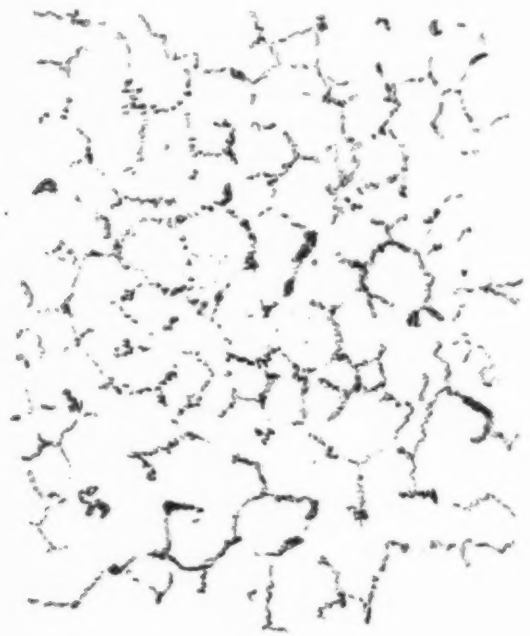

FIG. 5.—Surface of peritoneal epithelium, showing mode of distribution of pigment injected into the peritoneum.

On the surface of the epithelial cell layer the pigment is scattered abundantly in fine granules but when we examine such a surface with the microscope it is found that the distribution is not a completely irregular one but that as shown in Fig. 5, the granules are ranged along the outlines of the epithelial cells in such a way as to roughly imitate the picture produced by treating the peritoneum with silver nitrate. It is evident then that instead of pouring in a vortex into a single hole or a series of large holes in the diaphragm the pigment granules press through between each two cells and indeed all along the margin of each cell—in every part of each intercellular space we find them. In section it is seen that these granules often form little masses which lie on the basement membrane between the cells, and granules are even found running in long lines beneath the cells and between them and the basement membrane.

Evidently these epithelial cells form a very slight obstruction indeed to the entrance of pigment. Often it is found even to extend into the crevices of the connective tissue away from the lacunæ but it is more especially into the lacunæ themselves that its wandering may be traced. (Fig. 6.) Here, as it will be remembered, we find at most a lattice work of fibres between the epithelial and endothelial layers and through this and among the underlying connective tissue fibrils the granules may be traced down to the endothelium. Sometimes they are arranged singly and in small groups which adhere to the connective tissue fibrils and outline them while at other times they are packed together in a dense mass which extends through the tissue and more than anything else suggests the idea of the wide open stomata packed with pigment. These pigment granules may run along the surface of the endothelium or bulge it into the lumen of the lymphatics and where there are also masses within the lumen it is difficult or impossible to determine the line of the endothelium. The exact anatomical relations are thus very much masked by the presence of so much pigment, but from the knowledge of the anatomy of the tissues which we have gained, we must believe that in all these cases the pigment masses without the lymphatic are continuous with those within the lumen through the intercellular spaces between the endothelial cells, produced by a relaxation of the connection of these cells with one another, a separation which is increased by the greater and greater accumulation of pigment. In every case the pigment comes to lie directly upon the endothelium and as may be seen when such specimens are injected with silver nitrate, becomes scattered over the surface, the granules being most abundant along the margins of the endothelial cells, although they are by no means so abundant as to outline these cells. They show no especial relation to any accidental silver rings which may occur. To explain the passage of these granules through the endothelial wall into the lumen of the lacuna it seems necessary to suppose that the connections of the endothelial cells are so lax that the violent pumping action of the respiratory movement is enough to force material between them when they come to form the only obstruction to its entrance.

In reality, as is evident, this is very near practically to v. Recklinghausen's idea of stomata but it differs in holding the peritoneal epithelium as a complete layer of cells distinct from the lymphatic endothelium and not passing over into them through any communicating channel. It admits however a laxity in the connection of these cells with one another which allows of all the functional possibilities provided for by v. Recklinghausen's lymphatics in open communication with "saftkanälchen" or lymph canaliculi.

We have therefore in the absorption from the peritoneum a process in which a very important role is played by the phagocytic cells but in which the mechanical aspiration

FIG. 4.—Roof of a lacuna in section showing the entry of pigment by the aid of phagocytic leucocytes. Epithelial and endothelial cells are also phagocytic.

FIG. 6.—Roof of a lacuna in section showing entry of free pigment between the cells. Cross sections of the lattice work of fibres are seen in the roof of the lacuna.

of granules through the lax cell membranes by the respiratory movements is also possible.

Efforts were made to confirm these ideas or at least to shed additional light upon them by the study of the course of development of the diaphragmatic lymphatics and of the peritoneal cavity in embryo pigs. It was found practically impossible to inject the lymphatics of the diaphragm in very young pigs or indeed in any embryos from the thoracic duct, apparently because of the intervening lymphatic glands. The earliest stage in which an injection could be obtained by direct puncture of the diaphragm was that of 40 mm. but by staining the whole diaphragm in nitrate of silver (½% solution) it is possible to demonstrate endothelium lined lymphatics in the pig of 35 mm. Now the peritoneal cavity is fully formed and separated from the thoracic by the diaphragm at an earlier period than this, so that it seems entirely probable that the lymphatics have secondarily grown into the diaphragm from its edge and have later established the intimate relationship with the peritoneum described above.

From these studies then, we find no support whatever for the statement that there exist open communications between the peritoneum and the lymphatics, nor can we uphold the idea that the peritoneal cavity forms a part of the lymphatic system. Each of these cavities is lined with cells which retain their specificity throughout and nowhere merge into one another or into the adjacent connective tissue cells. Each develops independently and it is only later that they come into such intimate relationship that the transfer of materials from one to the other is relatively easy. The idea of the complete specificity of the endothelial cells of the lymphatics was expressed also in the previous paper mentioned and has since been confirmed—indeed one might say proven—by the embryological investigations of Dr. Sabin. And it does not interfere with the truth of these statements, that we find that the individual cells in any of these membranes are separable by adequate forces and that the mechanical violence of the respiratory movements is sufficient to force fine granules between them.

ON THE OCCURRENCE OF DIASTOLIC MURMURS WITHOUT LESIONS OF THE AORTIC OR PULMONARY VALVES.

BY RICHARD C. CABOT, M. D., AND EDWIN A. LOCKE, M. D., of Boston.

Two years ago we made a study of the clinical records and autopsy findings of all the cases which had died at the Massachusetts General Hospital with signs supposed to be due to valvular heart disease.

The chief object in this study was to determine in what valvular lesions diagnosis is most frequently correct, so far as autopsy can be made a test of correctness. In brief the results of this work were to show that in this series (153 autopsies) *mistakes in diagnosis* were most frequent in tricuspid regurgitation and *least frequent in aortic regurgitation.* Although there were several cases in which a shrivelled aortic valve (unsuspected during life) was found at autopsy, there was not a single case in which a diagnosis of aortic regurgitation made during life failed to be substantiated at autopsy.

In view of these facts it was a matter of considerable surprise to us to see during the past year four cases in which, largely owing to the presence of a diastolic murmur, aortic regurgitation was suspected during life, but, at autopsy, normal aortic valves were found.

Brief summaries of these cases follow:

CASE I.—Uncompensated cardiac disease in an adult alcoholic. Heart slightly enlarged transversely. Visible arteries pulsate strongly. Pulse irregular, low tension. Systolic murmurs in aortic and mitral areas.

Diastolic murmur in 3d and 4th intercostal spaces, close to left sternal margin. Urine of passive congestion (apparently). Blood normal. Death 11 days after entrance.

Autopsy.—Cardiac valves and cavities normal. Aorta 8 cm. in circumference. Hypertrophy of left ventricle. Chronic interstitial nephritis. Other organs not remarkable.

CASE II.—Three weeks orthopnœa with œdema of legs and face in a very anæmic woman 20 years old (scarlatina in childhood). Red cells 2,800,000, hæmoglobin 20 per cent. Marked transverse enlargement of heart. Pulse 120, regular. *high tension. Diastolic murmur loudest over 4th and 5th left costal cartilages.* Urine 1460 cc., 1009 in specific gravity. Albumen ⅛ per cent. No casts.

Autopsy.—Hypertrophy and dilatation of the heart. *No valvular lesion.* Aortic valve 5.5 cm. in circumference. Kidneys show lesions of chronic glomerulo-nephritis. Other organs not remarkable.

CASE III.—Woman, 34, alcoholic, very anæmic from profuse and frequent nose-bleed and from a recent post partum hemorrhage. Red cells 840,800. Slight transverse enlargement of the heart. *Diastolic murmur loudest over 4th left costal cartilage.* Systolic murmurs at aorta and mitral areas. "Flint" murmur at apex. Corrigan pulse, low tension. Urine of diffuse nephritis. Died in 3 days.

Autopsy.—Fatty heart; valves and cavities normal. Fatty kidneys. Yellow marrow (fatty).

CASE IV.—Typical pernicious anæmia. At entrance (after exertion) a diastolic murmur along left sternal margin. Later this murmur disappeared. Death four months later.

Autopsy.—Ordinary lesion of pernicious anæmia. Heart fatty. Valves and cavities normal.

CONDITIONS AND OCCURRENCE.

Diastolic murmurs, aside from those due to injury or deformity of the aortic or pulmonary valves, have been known to occur under each of the following conditions:

1. In association with dilatation of the aorta.
2. In association with intense anæmia.
3. In association with tuberculosis of the lungs and pleura, and in other conditions involving an abnormal suction or pulsion exerted by the heart upon neighboring portions of pulmonary tissue ("cardio-respiratory murmurs").
4. As an accentuation of a venous hum transmitted from the neck veins or produced in the vena cava superior.
5. In association with mitral disease and dilatation of or hyper-pressure in the pulmonary artery.

Only the first three of these conditions will be discussed in this paper.

We find no mention of diastolic murmurs without valvular lesions or aortic dilatation in the text-books of Osler, Musser, Tyson, Anders, Da Costa, Wood and Fitz, The Twentieth Century Practice, Loomis-Thompson, Allbutt, Gibson, Broadbent, Bramwell, Strümpell or Charcot and Bouchard.

I. *Diastolic Murmurs supposed to be due to Stretching of the Aortic Ring.*

This class of cases has been long known and is mentioned in most modern text-books. Corrigan[1] was apparently the first to maintain that if the aortic ring was stretched or dilated as the result of aneurism or diffuse dilatation of the arch, a genuine regurgitation of blood might occur with intact aortic valves. Two cases with autopsy are adduced in support of his belief, in which the valves were "nearly quite sound" but in which a double murmur (systolic and diastolic) was audible over the base of the heart and over the larger arteries springing from it.

Aran[2] mentions a similar case: "Les valvules sont épaissies et presentent des légères indurations à leur base, mais elles sont mobiles." The aortic ring was dilated to "deux pouces, dix lignes de circonference."

Besnier[7] heard a diastolic murmur and suspected aortic regurgitation in a case presenting at autopsy "une légère dilatation uniforme et regulier de l'aorte à sa sortie du péricarde; elle n'offre ni plaques calcaires ni atherome." The aortic valves were "minces, souples, lisses, sans adhérence ni induration."

Similar cases were recorded by Bellingham,[8] Peacock,[9] Perls,[9] Guttman,[9] Cockle,[9] Pel,[10] Chauffard,[11] Finlayson,[12] Massalongo,[13] Bouveret,[14] Renvers,[15] Jacquet,[16] Dombrowski,[17] Bonnett,[18] Pitt (8 cases),[19] and Klein.[20] [See also a discussion in which Farbringer, Leyden, Gerhardt, Litten and others took part.]

In Finlayson's and Renvers' cases the aortic valves were sufficient to the water test post mortem. In Dombrowski's case the aortic orifice measured nearly 9 centimeters. (In Bouveret's third case 8 centimeters.) In the last-mentioned case there was some atrophy of the kidneys (115 and 140 grammes) with increased arterial tension, and to this cause Bouveret refers the dilatation of the aortic orifice.

Yet as regards the occurrence of relative aortic insufficiency of this type there are some doubters, e. g., Friedreich,[22] Rosenstein,[23] Peter[24] and Potain.[25]

For the water test, on which many authors rely for proof of aortic insufficiency, is, of course, not regarded as final by modern pathologists, and furthermore, few of the writers who describe relative aortic insufficiency secondary to a sclerosed and dilated aorta have described the aortic valves as *entirely* sound. Experimentally it has been found very difficult to produce any considerable dilatation of the tough aortic ring even by exerting upon it a pressure much higher than that occurring in arterio-sclerosis and chronic nephritis.

II. *Diastolic Murmurs Associated with Intense Anæmia.*

Gerhardt,[26] Scheube,[27] O. Vierordt,[28] H. Vierordt,[29] Eichhorst,[30] and others, mention "accidental" or "anæmic" diastolic murmurs as possible, but very rare.

Litten (Deut. med. Woch., Feb. 24, 1885) refers very briefly to the occurrence of diastolic murmurs in "anæmic, poorly nourished" women, the murmurs being independent of any demonstrable lesion of the heart or aorta, and not due to venous murmurs transmitted down from the neck.

Klein[31] reports a case of extreme anæmia in a girl suffering from chronic Bright's disease. In the "aortic area" there was heard a diastolic murmur following the second sound, and the diagnosis of aortic regurgitation was made. At autopsy the valves were normal, the aorta not dilated, the lungs normal.

V. Noorden[32] records "presystolic" (not diastolic) murmurs in 2 cases of intense anæmia with no valvular lesions post mortem. A similar case has been observed by one of us.

Sahli,[33] in discussion of what he calls "Functional diastolic heart murmurs," concludes that when blood pressure is high, there may be enough dilatation (temporary or permanent) at the aortic and pulmonary orifices to prevent the closure of these valves.

He also discusses and exemplifies what he calls "accidental diastolic heart murmurs" in two cases of grave anæmia (red cells 720,000 and 796,000) precisely like some of those which we shall report below. In these cases no dilatation of the aorta could be made out post mortem and the valves were intact, but the left ventricle was in both cases dilated. The diastolic murmur appeared for the first time a few days before death. These murmurs, Sahli believes, to be independent, not only of any valvular lesion, but of any temporary stretching of the aortic orifice, for such a dilatation must depend (according to him) upon high blood pressure in the great arteries, while in the two cases mentioned the pressure appears to be notably low. Sahli believes that the murmurs were due in these cases to the extreme thinness of the blood.

In both cases there were also present venous murmurs in the neck, but their pitch, duration and point of maximum intensity were quite different from those of the murmurs in ques-

tion, and between the two points of maximum intensity there was a zone free from any adventitious sound whatever.

III. *Cardio-Respiratory Diastolic Murmurs.*

No attempt has been made to collect all the literature of this phase of our subject, but examples of German, French, English and American writings are subjoined.

Friedreich [4] mentions as a great rarity a case of phthisis "with anæmia" in which during life he heard a musical diastolic murmur over the left ventricle. There was found bilateral pulmonary tuberculosis post mortem and the conditions seem to have been favorable for the production of cardio-respiratory murmurs, though Friedreich suggests no such explanation.

Weiss [22] reports three cases with diastolic murmurs along the left border of the sternum, loudest in the third, fourth and fifth spaces respectively. No anæmia and no dilatation of the aorta is mentioned, but in all three cases there was extensive pulmonary tuberculosis. It seems possible that the murmurs may have been of the cardio-respiratory type, but they persisted in all phases of natural, forced and arrested breathing. There were no murmurs audible in the neck-veins.

Potain [23] reports three cases apparently similar to the above, but without autopsy, one case with autopsy at which tuberculosis of the peritoneum, lung and pleura was found, and another with autopsy and no tuberculosis. In one of the cases without autopsy the murmur disappeared when the patient sat up.

Drummond [27] records two cases of phthisis with diastolic murmurs disappearing on holding the breath.

Hoover [28] says in discussing cardio-respiratory murmurs that they "rarely accompany sharply the diastolic tone, but more often directly follow it." He describes two cases, in one of which there occurred a diastolic murmur audible at first in the second right intercostal space and at the apex, but later over the whole præcordia. The heart was dilated and insufficient. The murmur persisted in all the phases of natural and forced respiration and could not be made to disappear by changing the position of the patient. There were no sounds audible over the jugular veins nor over the bulbus venosus. Hence, Hoover made the diagnosis of aortic regurgitation, but "the autopsy revealed not the slightest sign of an endocarditis at any point, nor were there any evidences of dilatation of the aorta or of the aortic ring."

In Hoover's second case the diastolic murmur was in the pulmonary area and could be made to disappear on forced expiration.

In the first case then there was nothing adduced as evidence to show that the murmur was of cardio-respiratory origin, and we are inclined to believe that it was of the same nature as one of those which we have ourselves observed.

DISCUSSION OF THE WRITERS' CASES IN THE LIGHT OF THOSE PREVIOUSLY REPORTED.

The first of our cases very possibly belongs to the group of those in which the diastolic murmur is supposed by most writers to be due to a genuine regurgitation at the aortic orifice resulting from dilatation of the ring into which the valves are inserted.

The other three cases appear to belong to the class of diastolic murmurs studied especially by Sahli,[26] and supposed by him to be due to the *intense anæmia* present in his cases as in ours.

Two explanations of these murmurs seem most plausible:

1. That of Sahli, that they result in some way from the extreme thinness of the blood.

2. That they are due to a *temporary* elastic stretching of the aorta, relieved when the heart ceases to beat, and therefore not demonstrable post mortem.

Against the second of these explanations Sahli urges the powerful objection that the blood pressure in his cases was very low and that high blood pressures would be necessary to produce any stretching of the aortic ring. Low blood pressure was present in two of our three anæmic cases; in the third, owing to a chronic glomerulo-nephritis the blood pressure was high and might conceivably have stretched the aortic ring, especially as the aorta was unusually thin and elastic in this, as in so many cases of intense anæmia.

For the other two cases we have no better explanation than Sahli's to offer (extreme thinning of the blood), though this does not seem to us by any means a satisfactory one. There was no venous hum in any of our cases, nor was any marked change produced in the murmurs by changes of position or by the different phases of respiration.

Details of our cases follow:

CASE I.—James Coleman, a barber of 50, entered the Long Island Hospital July 9, 1900. His family history and past history are not significant. He had used whisky to excess for 20 years. His present complaint is of dyspnœa, muscular weakness, and swelling of the feet, symptoms which first showed themselves three months ago and compelled him to give up work two months ago.

Physical Examination.—The heart's impulse, which was diffuse, could be seen and felt and extended half an inch beyond the nipple in the fifth interspace. The right border extended half an inch beyond the right edge of the sternum. The action was very irregular. A soft systolic murmur limited to the region of the apex beat and another systolic murmur at the second right interspace were easily audible, the latter only after exertion.

In the 3d and 4th intercostal spaces just to the left of the sternum, a diastolic murmur was heard.

The pulse was of low tension, irregular and intermittent. Visible pulsation was noted in all the superficial arteries. Moderate œdema of the lower legs. All the superficial arteries were tortuous, stiff and uneven of surface. At the base

of each lung behind there were a few crackling râles. Physical examination was otherwise negative.

Blood showed red corpuscles 4,200,000; white corpuscles, 5600. Urine, high in color, acid, 1020, ⅛ per cent alb., no bile or sugar. The sediment contained a little abnormal blood and a few hyaline granular casts.

The patient lived but six days after entering the hospital and did not react at all to treatment. The clinical diagnosis ante mortem was aortic regurgitation, mitral regurgitation, myocarditis, chronic interstitial nephritis.

Autopsy.—July 21, 1900. Was performed by Dr. Geo. B. Magrath, and may be summarized as follows:

Heart hypertrophied; weight 480 grams; wall of left ventricle 20 mm. in thickness. The valves and cavities normal. The aortic valve 8 cm. in circumference, the others within normal limits. Coronary arteries present a moderate degree of arterio-sclerosis.

Pleural cavities both partially obliterated by old fibrous adhesions. Lungs show considerable œdema and congestion. Peritoneal cavity contains about a pint of turbid straw-colored fluid.

Kidneys combined weight 260 grams. Capsule strips with some difficulty, leaving a roughened surface. Cortex .3 cm. in thickness; markings fairly distinct. The other organs presented nothing markedly abnormal.

Case II.—Mrs. Tuttle, 20 years old (married), had scarlet fever in childhood, but apparently recovered her health and remained well until 1899, when she had an attack of "rheumatism around the heart." Had a miscarriage about this time, but has never had a living child.

Three weeks before entrance, dyspnœa, orthopnœa, cough with blood-streaked sputa and œdema of legs and face began to distress her and for relief of these symptoms she entered the Samaritan Hospital Sept. 21, 1901, in the service of Dr. E. P. Joslin, who has very kindly allowed us to use the case.

Examination showed a very small and pale woman (weight 90 lbs.), with orthopnœa and a pulse of 132, of small volume and low tension. The heart's apex in the 5th interspace, 5 cm. outside the nipple. There was a systolic murmur and thrill most marked in the third left interspace outside the nipple, but also noticed over the whole præcordia and in the left axilla. Slight œdema of extremities and of the bases of the lung.

Blood: Red cells 2,860,000, hæmoglobin 20 per cent.

Urine: 450 cc. in 24 hours. Sp. gravity 1011. A large trace of albumen; rare hyaline and granular casts.

Sept. 27.—Loud blowing diastolic murmur in the 4th left interspace. Presystolic thrill and rolling presystolic murmur in the second and third left interspaces. The first sound at the apex very sharp, the second sound there also loud and ringing, as was the aortic second.

The patient was seen Oct. 13 by one of us who made the following record: "Most comfortable in a semi-reclining position. A space the size of the palm in the region of the left nipple rises with each systole. The apex beat extends 2 inches beyond the center of the nipple in the fifth space.

"Cardio-hepatic angle (by percussion) acute. Cardiac dullness 2½ cm. beyond the right sternal margin.

"Both sounds at apex clear and strong, the second accompanied and followed by a short, high-pitched murmur occupying the first two-thirds of diastole and heard loudest over the 4th and 5th left costal cartilages. Pulmonary second sound accentuated and reduplicated. In this area the diastolic murmur has at times a musical quality. Aortic second weaker than normal.

"Pulse regular; 120; the wave rather sharp at the summit and collapsing somewhat more rapidly than normal, but it is *difficult to compress it.*

"Bubbling râles at the base of both lungs."

Urine 1460 cc. in 24 hours; 1009 in sp. gravity; albumen ⅛ per cent; no casts. Red cells 3,308,000. Hæmoglobin 40 per cent.

Nov. 2.—Tic-tac rhythm; systolic tricuspid murmur; no diastolic murmur heard. Right border reaches half way to right nipple (percussion).

Nov. 14.—Apex in anterior axillary line. Diastolic murmur faint, but unmistakable. Second sounds at base strong. Slight ascites.

Urine 840 cc.; albumen ⅛ per cent. Few hyaline and granular casts.

Died November 18.

Autopsy (Dr. J. H. Wright).—Heart showed no valvular lesion. Marked hypertrophy and dilatation (weight 525 grams). Aortic valve 5½ cm. in circumference.

Kidneys: Combined weight 68 grams; 7.3 and 7.6 cm. in length. Microscopically they showed the lesions of chronic glomerulo-nephritis.

The other organs not remarkable.

Case III.—Annie McDonald, a married woman of 34, entered the Massachusetts General Hospital February 6, 1902. Nothing of importance in the family history or past history of the patient, except that she had used alcohol to excess. For two years she has been troubled with œdema of the legs, and frequent nose-bleed. For a year dyspnœa, palpitation, extreme weakness and pallor. For three months her face has been puffy in the morning and she has had to pass urine several times every night. The nose-bleed above referred to came at first every week and often lasted five or six hours.

Three weeks ago she was delivered at the Lying-in Hospital of a full-term child, after a normal labor. Following it she has had a series of very profuse uterine hemorrhages, resulting in extreme anæmia and prostration. At the time of entrance the patient had scarcely strength to lift her hands, was at times delirious and had been vomiting so persistently that rectal feeding had to be adopted.

Physical Examination.—Skin very pale yellow; no emaciation. By percussion and palpation the heart's impulse was found to extend 16 cm. from the mid-sternal line in the fifth intercostal space. No enlargement could be made out to the

right. Action strong and regular, impulse heaving. Over the whole præcordia, but with greatest intensity over the 3d and 4th left costal cartilages, a blowing, diastolic murmur was heard. Systolic murmurs were heard in the aortic area and at the apex, the former transmitted to the neck, the latter transmitted to the axilla and to all parts of the front of the chest. Over a small area near the apex impulse in the nipple line was a well-marked palpable thrill and presystolic rolling murmur. Pulmonic second accentuated, aortic second scarcely audible. The pulses equal, synchronous, of large volume and low tension ("Corrigan pulse"). Capillary pulse not visible. No abnormal sounds in peripheral arteries. Lungs and other organs negative.

Red corpuscles, 840,800; white corpuscles, 6300; hæmoglobin, 15 per cent. Differential count of 400 white cells showed: polynuclears, 57 per cent; lymphocytes, 42 per cent; eosinophiles, 1 per cent. While counting these, 6 normoblasts and two megaloblasts were seen. There was moderate poikilocytosis with slight achromia and a tendency to small size.

Urine: pale, acid, 1014, alb. sl. poss. tr., no bile or sugar, urea, 1.39 per cent. Sediment: many fatty renal cells, occasional hyaline and finely granular casts with fat and renal cells adherent.

Patient failed rapidly and died in three days.

Autopsy, Feb. 9 (Dr. J. H. Wright), showed, scattered over the face, neck and trunk and extremities, a number of purplish red spots, largest about 3 mm. in diameter. About 200 cc. of serous fluid in pericardium. Ecchymoses in epicardium of both ventricles. Heart of normal size, weight 338 grams, valves and cavities normal. The myocardium firm, brownish, the papillary muscles covered with a fine yellowish mottling. Microscopical examination of a teased preparation from the myocardium of the left ventricle showed a moderate amount of fatty degeneration of the muscle fibers. Kidneys: combined weight 294 grams. Capsules not adherent. Cortices of normal width and showing the normal markings. Substance of kidneys more resistant to touch than normal. Pale, brownish gray in color and rather translucent.

Microscopical examination of a frozen section shows degeneration of many tubules.

The uterus almost filled with a mass of clotted blood. The mucous membrane of the stomach showed a moderate number of scattered red points and a thin linear cicatrix about 2½ cm. long near the lesser curvature.

The marrow of several vertebræ and of the shaft of the femur was for the most part yellow, and apparently fatty. The other organs showed nothing remarkable. Cultures negative.

CASE IV.—John W. Cole, sea-captain of 56, entered the Massachusetts General Hospital December 21, 1901. There was nothing remarkable about his family history, past history or habits. He entered the hospital on account of increasing dyspnœa, muscular weakness and pallor, symptoms which have been noticed for about four months. A little puffiness of the face and ankles has been noticed at times. He is thirsty but has little appetite and his food feels heavy in his stomach two or three hours after meals. He has had no nausea or vomiting. He has lost no flesh and had worked until he entered the hospital.

Physical Examination.—A well-nourished man with a pale lemon-yellow colored skin. The heart's impulse was palpable in the 5th space 12 cm. from the mid-sternal line. No enlargement to the right. The action was regular and of fair force. Along the left edge of the sternum was a faint blowing murmur occupying the whole of diastole. In the aortic and mitral areas there were soft systolic murmurs transmitted respectively to the neck and to the axilla. Pulmonic second slightly accentuated. Radials palpable, pulses synchronous, slightly irregular and poorly sustained, but not of the "Corrigan" type. No capillary pulse no abnormal sounds in the peripheral arteries.

Moderate œdema of the lower back, of the thighs, legs and genitals. Examination of the eye ground by Dr. Cheney showed retinal hemorrhages.

Red corpuscles, 904,000; white corpuscles, 5000; hæmoglobin, 25 per cent. Differential count of 400 cells showed: polynuclears, 65.2 per cent; lymphocytes, 34.8 per cent; eosinophiles, 0. While counting these, 6 megaloblasts and 2 normoblasts were seen. There was marked poikilocytosis with predominance of macrocytes and slight polychromatophilia.

Urine: normal color, acid, 1016, alb. sl. poss. tr., bile absent. Urea: 36 grams in 24 hours. Sediment not pathological.

The patient gradually failed from week to week and with the advance of the disease the diastolic murmur disappeared. The other murmurs persisted. There was no other change of any importance either in the blood or in any of the other signs. Death occurred the 29th of March.

Autopsy, March 30, 1902 (Dr. J. H. Wright), showed a large accumulation of fluid in each of the serous cavities and in the subcutaneous tissue. The heart weighed 370 grams; the wall of the left ventricle 12 mm. thick. Valves and cavities not remarkable. A moderate amount of fatty degeneration of the myocardium was shown on microscopic examination.

Kidneys combined weight 362 grams, capsule not adherent. Cortices of normal width and their markings normal. Consistency of the organ increased markedly and on section the tissue has a flint-colored appearance.

The mucous membrane of the stomach showed numerous red spots and points.

The marrow of the sternum of several vertebræ and of the shaft of the femur is transformed into a dark red, fleshy, friable, moist material. The other organs showed nothing of importance and cultures from the heart, liver and spleen showed no evidence of general infection.

Summary and Conclusions.

1. Diastolic murmurs without organic valve lesions are not uncommon in connection with dilatation of the aorta, localized or diffused. One of our cases seems to be of this type.

2. When the pleura and pericardium are adherent, owing to tuberculosis or other causes, diastolic murmurs are occasionally audible in the præcordia. Such murmurs are notably affected by respiration and by position; they are probably due, in most cases, to suction or pulsion exerted by the heart upon portions of lung adherent to the pericardium ("cardio-respiratory murmurs").

3. In cases of intense anæmia, when the red cells are reduced to or below 1,000,000 per cu. mm., one occasionally hears diastolic murmurs not to be explained by permanent dilatation of the aortic ring nor as "cardio-respiratory murmurs," and not due to a diastolic accentuation of a venous hum.

The cause of these murmurs is obscure.

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THE USE OF THE X-RAY IN SURGERY.[1]

By E. A. Codman, M. D., Boston.

Since I began practice in December, 1895, most of my experience has been obtained in the surgical wards of the Massachusetts General Hospital. It happened that in the very month in which I started practice the discovery of the X-ray was announced and within a few months I began using it. I took it up because it seemed to me likely to become a valuable aid in surgery and for the same reason I have followed its development since. What I am to say to you to-night, therefore, I trust you will take as from a surgeon rather than from an X-ray specialist. I hope that it is because I am a surgeon that I have the honor of speaking to you on the X-ray.

At the Massachusetts Hospital the X-ray is in daily, almost constant use. We are fortunate in having a skiagrapher whose sound sense and intelligent interpretation of the plates has made his opinion valuable. It is a significant tribute to the worth of the X-ray in diagnosis to see, that though this skiagrapher has no medical education as such, he is almost daily called upon to make diagnoses of the various forms of fracture or to differentiate between tubercular and syphilitic diseases of the bone, osteomyelitis and sarcoma. Probably our experience of the X-ray has been much the same as yours. I may sum it up thus: many disappointments, occasional successes, great hopes for the future. We find it especially useful in the diagnosis of fractures and bone diseases, in the localization of foreign bodies and concretions, and in the treatment of epithelioma, psoriasis and eczema.

[1] Read before the Johns Hopkins Medical Society, February 2, 1903.

Occasionally it enables us to demonstrate a suspected aneurism or to determine the exact size and position of the heart, but it is not yet of much practical value in the diagnosis of thoracic diseases.

I have seen trouble caused by searching for foreign bodies which the X-ray showed very nicely. I remember a bullet in the brain which was diligently searched for. Shortly afterwards at autopsy it was found just beneath the dura. I remember a stone in the kidney which proved to be a button on the back of a nightgown. A few years ago interesting children coming to the accident room with pennies in their stomachs were admitted to the hospital; now they are allowed to pass the pennies at their homes. Occasionally, however, it is strikingly useful. In one case I remember rescuing a child from a brilliant œsophagotomy by extracting a cent impacted in the portion of the œsophagus just behind the intersternal notch. By looking with the fluoroscope I was able to see the jaws of the forceps open and close over the coin. I am satisfied that in small children coins impacted in the œsophagus can generally be removed in this way or dislodged. Incidentally I cannot speak too highly of Dr. Coolidge's method of locating and removing foreign bodies impacted in the bronchi by introducing a urethroscope through a tracheotomy wound.

We use the X-ray as a routine method in fractures. I am personally thankful that my house officer experience was in the days before the X-ray. Our house officer of to-day must leave the diagnosis to the skiagrapher. It is not fair to the patient to learn fracture pathology in the old way. I do think, however, that our greatest help from the X-ray in fractures is in learning more accurately their pathology. We see now so many skiagraphs that we jump from the look of the broken limb to the probable position and cleavage of the fragments. Thus, before the X-ray is taken, I find myself making correct clinical diagnoses of fracture of the carpal scaphoid, of the os calcis, and of certain fissures of the external malleolus, which in the old days were classed as sprains. Fractures are set better now than they were in my day. This is especially noticeable in Colles fractures.

In the detection of stones in the kidney and ureters we have been disappointed. As yet we have not been able to keep the routine standard of excellence of the plates to a point clear enough to say positively "yes" or "no." "Yes" we can say occasionally, "no" rarely. On two occasions I have failed to locate in stout people small renal stones, which were later removed. Unless the patient is decidedly thin I should not have confidence in a negative diagnosis. I believe that the question of operation in such cases should be decided by the gravity of the symptoms or by a positive skiagraph which has been several times repeated. In the localization of ureteral stones the X-ray is especially helpful. Dr. J. W. Elliott had a brilliant case at the hospital in which a ureteral stone was located and easily and successfully removed.

Of all the cases which attend our clinic none seem so willing to keep their appointments, to wait patiently for hours, or to show themselves as those which are waiting for X-ray treatment. Perhaps there are still among you skeptics who doubt the efficiency of the X-ray as a therapeutic agent. Two years ago I was a skeptic myself and went out of my way to find philosophical and psychical excuses to explain the zeal of patients to return for treatment. I confess I cannot wholly rid myself of these excuses yet, but I have now seen some successes so brilliant that I cannot wonder that they arouse hope in the hopeless and that the poor wretch with recurrent cancer of the jaw fancies that faithful attendance on the X-ray machine will cure him as it has the man who sat next him with epithelioma of the nose.

The therapeutic X-ray work at the hospital is at present mainly done by Mr. Dodd and Dr. Vose, who are working on malignant disease for the Cancer Commission, and by Dr. Burns, who does the routine work of the skin department. Since their work will undoubtedly some day be reported in full, I will merely make a few general observations.

Though many cases of deep malignant disease have been faithfully exposed, few have essentially improved and none have been cured. There have been a few encouraging signs in some cases as relief from pain, gain in weight, shrinkage, softening, breaking down of the tumor, etc., but since these events occur in the course of untreated malignant disease the positive advantage of the X-ray can hardly be proved. On the other side, cases of epithelioma are brilliant. About a hundred have healed or are healing. Occasionally one is referred to the surgical department for excision. I am sorry to say that the present enthusiasm sends the case to the X-ray first, for I still believe that in most cases there is no Indian so good as a dead Indian. The responsibility of removal should remain with the surgeon, and he should refer it to the X-ray when contraindications to operation exist. Leaving you to look forward to the reports of Dr. Vose and Dr. Burns, I will briefly take up the more interesting of my private cases, for I do no X-ray work personally at the hospital.

Dr. J. C. Warren operated on an unusually hopeful case of cancer of the tongue in April, 1902. As far as could be seen the disease was entirely removed and the wounds healed well. I began the X-ray in May for prophylaxis and kept the floor of the mouth and sides of the neck of the patient on the edge of troublesome dermatitis until September, by which time the disease had so far recurred in the mouth and neck as to make the X-ray a mere nuisance.

Since October 4 I have treated a case of cancer of the breast, which for certain reasons had never been operated upon. I have kept the skin constantly exfoliating and at times blistered. The growth remains, so far as I can tell, practically the same.

Note 1.—This remains true on April 14, 1903.

In a breast case operated on by Dr. A. T. Cabot in October, 1901, there were seven spots of cutaneous recurrence on December 4, 1902, about the size of lemon seeds. These were in

the skin rather than below the skin, but were not ulcerated. At the present date five have entirely disappeared and two can only with doubt be recognized. I do not mention this case as even benefited, because of course with seven skin recurrences the disease must also have recurred in the deeper tissues. I merely want to say that in some cases it is possible to remove by the X-ray the recurrent skin nodules.

Note 2.—All seven spots have disappeared and have not returned. April 14, 1903.

I have exposed without success two cases of cancer of the penis. On one case the cancer was but a quarter of an inch in diameter, but was so obstinate to X-ray treatment that an amputation was done, as there were other smaller areas of a similar nature on the glans. Possibly with more persistent and vigorous treatment I should have succeeded better. The sections from this case are shown in Figs. 1 and 2.

If I had come to speak to you in October I should have said that the only cases of malignant disease in which I had seen unequivocal improvement from treatment with the X-ray were those of epithelioma and superficial post-operative recurrence. A year ago in an article on X-ray burns I stated that there was no good evidence that the deeper tissues were affected unless the skin overlying them also was injured. I should have used this as an *à priori* argument against probable improvement in subcutaneous malignant disease.

Since then, however, I have had a case which you may take as the exception which proves the rule. On November 10, 1902, Dr. J. C. Warren referred to me a patient with sarcoma of the sterno-clavicular region on whom he had just operated. A small incision had been made and a piece of the growth removed and found by Dr. Whitney to be a round-celled sarcoma.[2] The tumor proved to be inoperable and within a short time I began the X-ray treatment. At that time the tumor was $5 \times 2\frac{1}{2}$ inches. At this date it cannot be measured, for nothing remains but a slight thickening about and behind the clavicle. The weak point of this case is that when the tumor subsided a subluxation of the clavicle was revealed. This fact suggests that the periosteum may have been torn and have formed a callous. Then too, I have seen at the hospital other cases of sarcoma faithfully followed by the X-ray and yet steadily grow worse.

I wish I could take the time to tell you of other cases—one of two small areas of facial lupus which after six months' fight aided by curetting finally healed, only to recur again in two months; cases of psoriasis and of chronic eczema which have faded away as if by magic, and yet have recurred in other places; and two of interesting, though sad, cases of lympho-sarcoma.

In speaking of X-ray therapeutics I have purposely avoided quotations from the writings of others, believing that what you want from me are merely the things I can swear to. You have read, as I have, certain rosy articles on the use of the X-ray in the more terrible forms of malignant disease, of the disappearance of sarcoma and cancer, but all I can tell you is that at the Massachusetts Hospital we do not get such good results and in my private practice I do not get such results. I have put my best foot foremost and told you of the most wonderful cases I have had, and later I will show you a lantern slide of the most successful hospital case.

However, I am ready to stand here and say to you that the effect of the X-ray in causing the disappearance of psoriasis, chronic eczema, lupus and superficial epithelioma is little short of the miraculous. In some cases I know that recurrence takes place, perhaps after too brief treatment. I believe, too, that though few reliable reports of success in deep cancer and sarcoma have yet appeared, considering the immense number of such cases which have been treated, the X-ray offers at least some hope and deserves an attention and dignity which has not yet been accorded it by the best of the profession.

I know that some of you will ask for a plausible theory of the action of X-rays. In answering this I will relax the strong effort I have made to stick to facts. It seems to me that the effects are due to stimulation of the nutrition of the parts rather than to destruction of the malignant tissue, because:

1. The effect often does not take place for days and sometimes for weeks.

2. From the analogy of so-called burns the primary effect is increased stimulation of the skin as evidenced by hyperæmia and exfoliation.

3. In one case of massive malignant disease of the breast which had been long and faithfully exposed no destruction of the cells was found. Degeneration in the interior of such cancers which have not been treated is often found.

4. The appearance sometimes found of shrunken cancer cells surrounded by granulation tissue may be explained by the activity of the tissue.

5. In a case of varicose eczema of the leg which I treated the eczema not only disappeared but the leg shrank to its normal size and its circulation became active.

6. The good effects occur chiefly in those diseases which are characterized by a disordered or indolent local nutrition, which nature when stimulated by other means has at least a chance of throwing off. For instance we all know that chronic eczema, psoriasis, lupus, rodent ulcer, epithelioma, even ulcerated cancer of the breast, may under improved conditions heal in whole or in part. Even in deep-seated

[2] At the operation on Mr. B at St. Margaret's Hospital, October 24, (S. 210-2), there was seen a diffused, infiltrating new growth over the clavicle which had grown into the muscle. The growth was white and fasciculated and to the eye presented the characteristics of a sarcoma.

Microscopic examination of the small fragment which was removed showed small, round cells in solid masses among which were occasional muscular fibres more or less degenerated, and widely separated from each other by the new growth. The cells of the new growth seemed to have a little rather homogeneous, and, in places fibrillated, tissue between them, and were traversed by very numerous, small blood vessels with distinct walls. Among the cells there were very numerous mitoses.

The structure of the growth confirms the diagnosis of sarcoma, and the type of cells places it in the round cell variety.

W. T. WHITNEY.

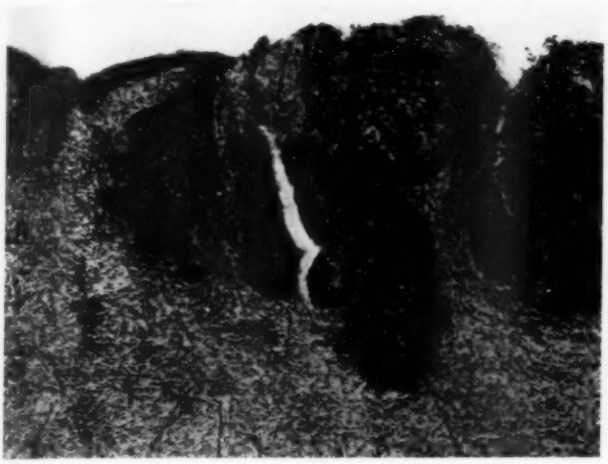

FIG. 1.

FIG. 2.

Sections taken from a case of cancer of the penis in which there were several distinct lesions, one of which had been treated much more vigorously than the others.

FIG. 1 was taken from a relatively untreated lesion and serves to show the type of cancer.

FIG. 2 was taken from the lesion which had been most treated and serves to illustrate the replacement of cancer cells by a granulation-like tissue.

Kindness of Dr. J. H. Wright.

FIG. 3.

FIG. 4.

A case of recurrent cancer of the breast before and after the use of the X-rays to illustrate the fact that though healing of a large part of the ulcerated portion has taken place, the subcutaneous growth in other places has continued to advance.

Kindness of Mr. Walter Dodd.

malignant disease we may find cases of spontaneous absorption of sarcoma, perhaps coincident with some outlandish treatment.

7. That of the above mentioned forms of disease nature is more likely to help in those in which the X-ray is most efficacious, while in cases where the malignancy of the cancer growth is great even the X-ray cannot check it.

8. That good and bad effects alike occur chiefly in the skin, the nutrition of which seems in a more delicate balance than that of the deeper tissues.

I mention my choice of aiding nutrition rather than causing destruction because our choice indicates our rationale of treatment. With any modern X-ray apparatus it is possible to cause necrosis of the tissues by decreasing the distance of the tube and prolonging the time of exposure. If we are doing good by destroying abnormal tissue we want to give large doses. If we aim to stimulate we should limit our doses to their physiological effects.

My method is to expose for ten minutes at ten inches as a standard dose. To repeat this twice a week until hyperæmia appears or we reach the end of the third week. By this means, the accumulated dose at the end of the third week only reaches 60 minutes at 10 inches. Then, if necessary, to increase it, never allowing a burn of the second degree if it can be avoided, but keeping the parts hyperæmic, pigmented or exfoliating.

Since other conditions being the same the intensity varies inversely as the square of the distance from the anode, we may reduce all exposures to this unit dose by using the formula:

$$\frac{T}{D^2} \times 100 = \text{the equivalent time at a distance of 10 inches.}$$

This, of course, implies keeping the tube at a high degree of efficiency, which for most good apparatuses does not greatly differ. In most individuals slight dermatitis usually appears in from two to three weeks with my apparatus, a Kinraide coil. In one case I caused a slight dermatitis by an exposure equivalent to 15 minutes, but usually it requires 40 to 100 minutes at ten inches in added exposures. Before reaching this point psoriasis and eczema may disappear, but in my experience the tide turns in epithelioma at about the same time as dermatitis appears. To cases which do not show at least slight improvement in six weeks I should give a bad prognosis. I do not believe that it is yet justifiable to use the destructive power of X-ray to cause necrosis in the manner of the old-fashioned cancer pastes, etc. The choice between such an X-ray burn and cancer is too terrible.

Finally let us remember that cancer may arise in an X-ray burn. Two such instances are on record already and one sad case has recently occurred in Boston. Cancer appeared simultaneously in the ring fingers of both hands, necessitating amputation at the first phalanges. The hands had been in a chronic state of ulceration from an X-ray burn for several years.

What information can we expect from the X-ray in cases of bone disease? This question may be partially answered *à priori* from a study of the physics of the X-ray. We cannot expect it to tell us the odor, color, sensitiveness, pain, temperature of the affected limb. No more can we expect it to give us a picture of the surface of the affected bone or of its cross or sagittal section. A skiagraph is a chart of the relative densities of the different portions of an object. More exactly it is a chart of the relative densities of substances encountered by the X-rays in their direct paths from the focal point on the anode to the photographic plate. The opacity of a substance to the X-ray varies directly with its atomic weight. Most errors in X-ray interpretation are made by looking at the skiagraph as at a cross section or as at a surface picture like a photograph. The outline is often the same, but in the skiagraph it bounds a chart of densities; in the photograph merely a picture of the surface. Therefore let us put it roughly, "Don't find fault with a skiagraph because it doesn't tell you how a bone smells."

What is the reason that pictures of the bones of the trunk in stout people are less satisfactory than of the limbs? Probably there is a little diffusion of X-ray light, but the main reason is that there is but a slight difference between the density of the total amount of substance encountered by the rays that only go through soft parts and those which go through both soft parts and bone. The contrast will stand in nearly the same ratio as the weights of equal columns of flesh to one of which a pinch of bone salts has been added. This is further complicated by other technical details as the nearness of the bone to the plate, the distance of the tube, etc. In some cases the contrast is so slight that we get no detail whatever and we must be content with merely knowing whether the bone is affected at all. In clearer pictures we may draw more complicated conclusions, e. g., that loss of substance has occurred, that new bone has been formed, that an old area of disease has become surrounded by new bone, that cavities in the bone exist, that sequestra have become isolated, and the like.

In fact we may infer with great exactness what the true pathological anatomy of the bone itself is, if we consider only the inorganic constituents of the bone. We may infer what its appearance would be if made into a dried museum specimen. But when we begin to infer the description of the organic elements we begin to make our mistakes. When we prophesy the contents of a cavity in the bone we must choose between many different forms of tissue with small atomic weight. Its contents may be cystic fluid, cartilage, pus, sarcoma, carcinoma, myeloma, gumma, osteoid tissue, tubercular granulations, or what not. In the same way cortical hypertrophy may be due to the poisons of phosphorus, syphilis, typhoid, tuberculosis, acromegaly, Paget's disease, osteo-arthropathie pneumonique and other conditions. Fortunately, however, there are often in skiagraphs certain finer details which may point more or less definitely to which one of these conditions has caused the formation of cavities or cortical thickening. For example the bone blisters in typhoid, syphilis and leprosy are not found in other diseases causing

cortical enlargement. Bone abscesses, gumma, periosteal sarcoma usually show the appearance of a cavity with thick walls, while myelogenous sarcoma, myeloma, bone cysts and carcinoma show an apparent cavity with thin walls.

As another instance some of the following points might aid us in the diagnosis of a diseased leg.

In syphilis increase of new bone is the rule; in tuberculosis destruction of old healthy bone. Syphilis rarely causes the formation of a sequestrum. Tubercular sequestra are always small and poorly defined. The sequestra of osteomyelitis are generally large, well defined and resemble the cortex of the old bone. Tuberculosis almost always starts in the epiphyses and is very rare in the shaft. Syphilis is almost always in the shaft and extremely rare in the epiphysis. Cortical thickening in syphilis is often very symmetrical, while in osteomyelitis it is irregular and fantastic. In syphilis the other unsuspected long bones usually show cortical enlargement, while in osteomyelitis only one bone is the rule.

Instead of confusing you with further descriptions, however, I will show you some lantern slides, for after all practice in the interpretation of the plates is the main thing.

Discussion.

Dr. Bloodgood.—Dr. Codman has left one very little to say except to thank him for the delightful and interesting discussion of the value of the X-ray in diagnosis and treatment.

In regard to the X-ray treatment of superficial epithelioma of the skin we have but one specimen in the surgical laboratory, which previous to its excision had been treated for some months with a Röntgen ray. Clinically the tumor was situated on the right side of the lower lip, and was about 8 x 5 mm. in diameter; it was considered to be an epithelioma. It had been present a number of months previous to the use of the X-ray. The X-ray treatment was followed for six or eight months and then discontinued. Clinically very little change was observed in the tumor. It surely did not get any larger, perhaps it got a little smaller. The tumor consisted of a small area of superficial induration at the muco-cutaneous border of the lower lip, and at one point on the mucous membrane side there was a superficial ulceration. The duration of growth from onset to the time of excision was about two years.

I was not informed of the date of the last X-ray exposure, but it was within at least four months of the time of the excision of the tumor. The tumor was removed by Dr. Finney and sent to the laboratory. It consisted of a V-shaped piece of tissue about 1.5 cm. in diameter, surrounding a small, rather circumscribed tumor at the muco-cutaneous border of the lip. On making one gross section the usual naked eye appearance of a squamous epithelioma was not definitely present. The tissues looked more finely granular, and one did not see the definite dots and lines, white in color, which in the majority of epithelioma can easily be seen, and are due to large alveoli of large squamous epithelial cells.

The first section cut and stained showed a very interesting histological picture; one could see normal skin to either side of the section, between this and extending into the deeper tissue there was a zone of tissue resembling granulation tissue. This zone of granulation tissue was not covered by normal epidermis, but by a layer two to three cells thick, of large epithelial cells. In the granulation tissue beneath this thin epithelial layer one could not recognize definitely any epithelial cells. This section therefore seemed to demonstrate what Dr. Codman has said, that the X-ray stimulates the formation of granulation tissue, and that this granulation tissue in its growth destroys the nest of epithelial cells.

February 10, 1903: Since discussing this section before the Medical Society further sections of the tumor have been cut and stained. These sections differ from the one described in that they show a definite squamous cell epithelioma. The sections, however, differ from the ordinary epithelioma in the great amount of granulation tissue between the epithelial alveoli. The first section seemed to demonstrate that perhaps the most of the epithelial alveoli had been destroyed, but further sections demonstrate that as yet the majority of the little tumor is a definite squamous cell epithelioma.

TRAUMATIC PERICARDITIS, ENDOCARDITIS AND MYOCARDITIS.

By J. Hall Pleasants, M. D., *Assistant in Medicine, Johns Hopkins University.*

While trauma as an occasional cause of cardiac disease is vaguely referred to in several text-books on medicine and surgery, the part which it may play in the production of pericarditis, endocarditis and myocarditis is not very generally appreciated. It should be understood that reference is here made not to direct injuries of the heart and its membranes by perforating wounds of the chest, but to the more indirect effect upon the heart and pericardium of contusions over the præcordia or elsewhere upon the thorax, or even as the result of concussion from severe blows upon distant parts of the body. Scattered throughout the literature several such cases have been reported. Two cases of pericarditis and one case of endocarditis, which have recently come to my attention, are of sufficient interest in this connection to report. The causal relation of trauma to these three cases seems unquestionable. In one of the cases of pericarditis there was also infarction of the myocardium, probably of traumatic origin.

Traumatic Pericarditis.

Case I.—July 7, 1902, I was called to see Mrs. ——, who had just been thrown from an open carriage. An examina-

tion showed severe contusions about the left shoulder and over the left scapular region. There was also a slight injury of the scalp, and the left radius was fractured. There was no sign of injury about the front of the chest. Considering the severity of the injuries, the general condition of the patient was good. She complained of considerable pain in the left arm, shoulder and back, but there was no pain about the chest. Careful examination did not show fracture of any of the ribs, although the pain under the left scapula pointed to the possibility of fracture of an underlying rib. *Examination of the heart and lungs was entirely negative.* Thirty hours after the accident the patient sent for me hurriedly, to say that a few minutes before she had felt a peculiar grating sensation under the sternum, which had lasted several minutes and then disappeared. A second examination of the heart and lungs was also negative. When I saw her the following morning she complained that the grating sensation had returned, and that she could now both hear and feel it. Examination over the chest showed the presence of a most intense to-and-fro pericardial friction rub on both palpation and auscultation. Nothing abnormal could be heard about the lungs, but later in the day a pleural friction rub was heard over the lower left back, which soon spread towards the axilla and over the lower left front. The loud pericardial friction rub continued to be heard over the entire præcordial region for one week, when it rapidly cleared up, and by the ninth day had entirely disappeared. About the same time the pleural signs also disappeared. At no time was there evidence of fluid in the pericardium or in the pleura. Over the præcordial region there were occasional sharp attacks of pain, usually lasting but a few minutes. With the exception of the pericardial friction rub and these occasional attacks of pain there were no other symptoms of pericarditis. The pulse was normal, and except for a rise of temperature to 101° on the second and third day, probably due to the external injuries, there was no fever. When last examined, six weeks after the accident, both heart and lungs seemed perfectly normal with no sign of pericardial or pleural adhesions.

In the following case the relation between the injury and pericarditis, found at autopsy, seems equally well established. The infarct in the myocardium appears to owe its origin to the same cause.

CASE II.—The patient, E. A. R., aged 55, worker in a brewery, was admitted to the Johns Hopkins Hospital in Dr. Osler's service October 3, 1902. Two days before admission he had been struck a severe blow in the left axilla with a piece of scantling, as it was being pushed through a partition by a fellow-workman. On admission he was suffering from severe pain in the lower left side especially on inspiration, shortness of breath, orthopnœa and cyanosis. Examination showed a large hyperresonant emphysematous chest. Pain on pressure in the lower left axilla indicated possible fracture of the ribs, which was later confirmed at autopsy. Everywhere over the chest sonorous and sibilant râles were heard, and in the lower left axilla the note was somewhat impaired. The heart was much enlarged, with no visible apex beat, the sounds being heard best in the 6th interspace 12 cm. from the mid-sternal line. There were gallop-rhythm and a loud systolic murmur at the apex. Pulse 112 to the minute. The urine contained albumin and casts.

The condition of the heart is especially interesting, as we have the notes of his physical examination just eighteen months before, when he was in the Hospital suffering from emphysema and chronic bronchitis. Then in addition to the typical signs of emphysema, he had an enlarged heart with the apex in the 6th interspace well out towards the axilla, and a loud systolic murmur. Nothing suggesting adherent pericardium was then noted. The principal etiological factors in his history had been alcohol and hard work.

Soon after admission his condition rapidly grew worse, the signs of heart weakness becoming more marked. On the fourth day a pleural friction rub in the lower left axilla and a pleuro-pericardial rub were heard. At the end of two weeks the pleural rub had disappeared, although the pleuro-pericardial rub was heard at intervals up to the time of his death, which occurred just four weeks after the injury. Although carefully looked for, neither a pericardial rub, fluid in the pericardium nor signs of adherent pericardium could be made out during life. Immediately following the injury there had been no fever except a brief rise to 100° on the third day. For several days preceding his death fever was present.

The autopsy notes by Dr. Marshall are of interest. The *anatomical diagnosis* was as follows: Fracture of the 6th, 7th and 8th ribs (left axilla), serous pleurisy; collapse of lower lobe of left lung; acute bronchitis; *subacute fibrinous pericarditis;* dilatation and hypertrophy of both auricles and ventricles; *infarct of myocardium;* chronic mitral endocarditis and insufficiency.

Thorax.—About 1500 cc. bright red turbid fluid in the left pleural cavity. Left lung: Adhesions between left lung and pericardium and a few old adhesions to diaphragmatic surface. Right lung: Adhesions between the lung and pericardium.

The heart with pericardium attached measures 14 cm. in widest part. The pericardium everywhere is closely attached to epicardium by *rather recent fibrous adhesions.* Mitral valve thickened considerably, irregular; no fresh vegetations; papillary muscles shortened; chordæ tendineæ thickened; aortic valve clear, with beginning calcification at root. Both left ventricle and left auricle dilated; myocardium rather opaque brown color; in one place entirely replaced by fibrous tissue. Coronary arteries normal.

The histological changes in the pericardium will be considered later. The infarct of the myocardium, which is about 1 cm. in diameter and situated in the wall of the left ventricle, will be discussed further when we consider traumatic myocarditis.

A brief analysis of these two cases emphasizes certain important facts.

In Case I the relation between the injury and inflammation

of the pericardium does not admit of any doubt. Examinations of the heart and lungs for the first twenty-four hours after the accident were negative. Then plastic pericarditis developed, followed by dry plastic pleurisy. While possibly the pleurisy was secondary to the pericarditis, it seems more probable that both developed independently as the result of the trauma, since the extension of such a non-infectious process as this, from the pericardium to the pleura, or the reverse, seems unlikely. The contusion in this case was over the left scapular region. The inflammation of the pericardium may have started in the posterior mediastinum. It seems possible, however, that the pericardium may have been injured by a *contre-coup* of the heart against the anterior chest wall. There is no reason to believe from the history of the case that inflammation was other than of a simple non-infectious type, such as is frequently seen in the synovia of joints as the result of mechanical injury.

In the second case the conditions are less simple. Here we are dealing with a diseased heart, that a year and a half previous was found to be much hypertrophied, with signs of relative mitral insufficiency. Following a severe blow, with fracture of three ribs in the lower left axilla, symptoms of defective heart action and myocarditis set in. Within four days after the injury a traumatic pleurisy, at first dry, but later with effusion, developed. A marked grade of emphysema of the lungs prevented the true condition of affairs in the pericardium from being made out clinically during life. The immediate cause of death seems to have been a terminal pneumonia four weeks after injury. The anatomical findings show a *recent* fibrinous pericarditis. The visceral and parietal layers of the pericardium are loosely bound together by recent fibrinous adhesions which are just becoming organized. From a microscopical examination of this partially organized exudate, the presence of numerous large fibroblasts, a rich supply of large capillaries, and areas of as yet unorganized fibrin, together with absence of any compact, well-organized fibrous connective tissue, and adult connective tissue cells, point to a process of not more than three or four weeks' duration. In the absence of cultures it is impossible to say with certainty whether the pericardial inflammation was of a simple or of an infectious type. From the stage to which it had advanced, it seems likely that bacterial infection had occurred, whatever may have been the character of the inflammation at the onset. Whether inflammation of the pericardium was secondary to the traumatic pleurisy, cannot be said with certainty, especially since it is not known whether the pleurisy was simple or infectious in character; nor is it known just when the inflammation of the pericardium began. Inflammation of the pleura and pericardium were both probably produced independently by the trauma. A blow in the left axilla would be transmitted with considerable force through a firm emphysematous lung against a much hypertrophied heart. The susceptibility of such a heart, already weakened by disease, to an acute inflammation would seem to be great. Although it cannot be said with absolute certainty that pericarditis in this case was not due to other contributing causes, the development of both pleurisy and pericarditis immediately after the trauma would seem to establish a causal relation with the injury beyond doubt.

With the exception of an occasional general statement in the text-books, that pericarditis may arise from injury, leaving us in the dark as to whether a perforating wound, laceration or contusion is meant, there are not many cases of traumatic pericarditis reported in detail. Billroth[1] has seen cases following left-sided fracture of the ribs, although no actual tear but only a contusion of the pericardium had occurred. He also saw one case following a blow upon the præcordia without fracture. Excluding cases caused by perforating wounds, I have been able to find only four cases in the literature reported in sufficient detail to draw any conclusions from them.

Golebiewski's Case.[2]—A workman aged thirty fell from a height of sixteen feet, fracturing the 6th rib in the left mammary line. Pericarditis with friction rub and some increase in cardiac dulness was first noticed about a month after the injury. There was dyspnœa and a very small, rapid and irregular pulse. At the end of three months the symptoms had entirely disappeared. There is no mention of an associated pleurisy.

Luckinger's Case.[3]—A man twenty-five years of age suffered a severe contusion over the left breast. There was no fracture of the ribs or sternum. Three days later palpitation of the heart, shortness of breath, cyanosis and fever developed. A pericardial rub with some effusion and a systolic and diastolic murmur were present. Later a slight circumscribed pleuritic effusion just below the apex of the heart appeared. The case is again referred to in the consideration of traumatic endocarditis.

Jessen's Case.[4]—A man, known to have been previously sound, fell, striking the left side of his chest against a piece of wood. There was no fracture of the ribs, but two days later evidence of slight tenderness over the 6th rib near the sternum. On the third day the cardiac dulness had extended out slightly both to the right and left; and over part of the body of the heart a fine friction rub developed. There was also a systolic murmur heard at times at the apex. On the sixth day he developed a rapid pulse, general weakness, shortness of breath and palpitation. The subsequent history is uncertain.

Thiem's Case.[5]—A man forty-eight years old received a severe blow on the left breast. There was fracture of the 6th rib between the mammary and the anterior axillary line. Four days after injury there was evidence of pericardial inflammation. Death occurred 16 days after injury. At autopsy no external injury was visible. There were subcutaneous emphysema and hæmorrhage into the intercostal muscles about the fracture. 500 cc. of turbid gray purulent fluid were found in the pericardium, with a thick layer of fibrin over the entire inner surface. The

[1] Billroth, Handbuch der allgem. und speciellen Chirurgie, Bd. iii.

[2] Golebiewski, Atlas and Epitome of Diseases Caused by Accidents. Phila., 1900, p. 208.

[3] Luckinger, Ueber traumatische Endocarditis. Münchener medizin. Wochenschr., 1892, Nr. 18.

[4] Jessen, Ein Fall von traumatischer Pericarditis. Monatschr. f. Unfallheilkunde, Leipz., 1898, 241.

[5] Thiem, Ein Fall von Quetschung- Herzbeutel- und Brustfellentzündung, idem, 1896, 65.

heart was somewhat enlarged with the heart muscle of a reddish brown color.*

Considering the frequency of chest injuries, comparatively few cases of traumatic pericarditis are observed. This is without doubt largely due to the frequency with which pericardial affections of all kinds are overlooked. Many of these cases come under the charge of the surgeons with the symptoms obscured by injuries of a more striking character or run a brief and mild course and thus readily escape notice. Out of a series of 100 cases of pericarditis in the Massachusetts General Hospital analyzed by Sears not a single traumatic case is recorded. Only one case (reported to-night) has been found among the records of the Johns Hopkins Hospital. That cases of this kind are as rare as would appear from this seems highly improbable.

The character of the injury is of interest. Perforating wounds, which for obvious reasons need not be considered here, may of course lead to direct infection of the pericardium. We are concerned now rather with the more indirect effect upon the heart of contusions about the thorax. Blows upon various parts of the chest can produce bruises and contusions of the surface of the heart with or without external evidence of injury. Suspended as the heart is in a sac within which it is quite freely movable, there is possibility of injury either by a direct blow where the heart is in close contact with the chest wall, or apparently as the result of *contre-coup* against the anterior chest wall from blows about the back. In one of my own cases the injury seems to have been by *contre-coup*. As far as I know this had not been pointed out as a possible cause of cardiac injury. As in Case I, there may be little or no evidence of external injury about the chest. Not only in pericarditis, but in traumatic endocarditis, considerable injury of the heart can take place without much evidence externally. There may or may not be an associated fracture of a rib. Of these six cases three show such an associated fracture of one or more ribs; in each of these cases the sixth rib, although in two instances it was fractured not over the pericardium, but well out towards the axilla. In the fatal case, where the injury was confined to the left axilla, the heart was much hypertrophied. That a heart can be injured even by severe concussion in a distant part of the body is shown in the case of reported rupture of a heart valve by a severe fall upon the buttocks. A study of the effects of trauma upon the heart, as shown by Riebold† and others in autopsies upon individuals who have died soon after severe thoracic injuries, throws much light upon the causation of traumatic pericarditis, myocarditis and endocarditis. In many cases we find actual bruising of the heart wall. In some cases it shows itself as a slight ecchymosis just under the visceral or parietal pericardium. There may be an actual tearing of the pericardium either with or without laceration of the adjacent muscle or effusion of blood into the pericardial sac. Such an injury may be the starting point of pericardial inflammation. The effect of contusions upon the endocardium and the myocardium will be referred to later.

The *type of inflammation*, like traumatic inflammation elsewhere, is simple, unless secondary bacterial infection occurs. Such invasion may occur through the blood current from an infected focus elsewhere in the body, by direct invasion from neighboring organs, or by extension from the adjacent pleuræ which have become infected. In Case II such a secondary infection with purulent effusion seems to have occurred. In three cases the inflammation was apparently simple. In the remaining cases we have not the data to decide whether or not secondary infection occurred.

The *character of the exudate* varies. It may be plastic in character, giving rise to a friction rub, as is seen in four of the cases, later going on to effusion as it did in three of these. Of the two cases which came to autopsy one showed general recent pericardial adhesions and the other a large (purulent?) effusion.

The *association of pericarditis with pleurisy* is interesting. Traumatic pleurisy is by no means uncommon and it also is usually simple in character unless the lung or chest wall is injured. When both pleurisy and pericarditis are of the simple type, the pleura and pericardium have probably both been injured independently, and there has been no extension of the inflammatory process from one sac to the other. That both the pericardium and left pleura should be so often affected together is not surprising, when we consider how difficult it would be to injure the pericardium without also involving the overlying pleura. The fact that pericarditis secondary to affections of the lungs and pleura is not uncommon in diseased conditions, is no argument for such an extension in simple non-infectious traumatic cases. Where there is an infection of either pleura as the result of a perforating wound of the chest or as the result of a laceration of the lung from trauma, a secondary infection of the pericardium can take place by direct extension. As concerns the relative frequency of traumatic inflammation of the pleura and pericardium I am inclined to think that, considering the total area of pleura and the area of pericardium exposed to contusions of the chest, pericarditis is probably of equal or greater proportionate frequency than pleurisy, owing to the less elastic character of the heart as compared to the lung, and therefore its greater liability to injuries by *contre-coup*.

The *symptoms* of these traumatic cases, as already seen, vary widely. In one of our cases, in which there was only a dry plastic exudate, with the exception of occasional pains about the heart, there were no symptoms. In the cases in which there was much effusion, the symptoms were those ordinarily seen in such conditions—disturbed pulse, palpitation, cyanosis and dyspnœa.

Fever seems to have been present in some of the cases. It

*In a case of traumatic endocarditis reported by Litten (Case V of summary) dry pericarditis developed on the eighth day after injury, and persisted "for a few days." The pericardial features of this case are very briefly discussed.

†Riebold, Bemerkungen über die Quetschung der Eingeweide von Brust- und Bauchhöhle. Friedreich's Blätter f. gerichtl. Medizin, 1890.

is often impossible to say whether it was due to the pericardial condition, or to other causes. Of our two cases, it was practically absent in one, and in the other, where it was present for two days, seems to have been due to other causes than pericarditis. The absence of fever points strongly to inflammation of a non-infectious type, in which there has been no secondary bacterial invasion.

Although the few cases here referred to would indicate that traumatic pericarditis usually runs a severe course, I feel sure this is misleading. It is more probable that the mild cases are generally overlooked, and the severe or fatal ones only are recognized and reported. A careful routine examination for cardiac involvement after thoracic injuries will doubtless reveal many mild cases like the first case here reported. Considering the frequency of injuries about the chest, a localized pericardial inflammation without other symptoms than perhaps a slight friction rub is probably of quite frequent occurrence. The "milk spots" upon the pericardium so often found at autopsy without a history of cardiac disease, are in some cases possibly of traumatic origin.

While pericarditis from injury may develop in a perfectly normal heart, an organ weakened by pre-existing myocardial disease would seem especially to favor its development. One of our cases well illustrates this liability.

It is unnecessary here to discuss traumatic pericarditis further. In the symptoms and physical signs, the more severe cases differ but little from the non-traumatic forms. In the more severe cases, especially where adhesions occur, the prognosis and course probably do not differ from the non-traumatic type. Some of the unexplainable cases of occasional sharp præcordial pain following accidents, may possibly be explained by the presence of a few localized pericardial adhesions of traumatic origin. That a slight exudate in the pericardium, or a localized plastic pericarditis of traumatic origin is probably overlooked, much more frequently than it is recognized, will scarcely be denied by those who are familiar with the discrepancy between the clinical recognition and the autopsy findings in non-traumatic pericarditis. A systematic examination of the heart in contusions about all portions of the thorax will alone give us an idea of its frequency.

Traumatic Endocarditis.

That acute or chronic endocarditis may develop in a heart previously sound as the result of injuries of the endocardium produced by contusions about the thorax, is even less generally known than is the occurrence of traumatic pericarditis. I am able to give you the clinical history of a case of traumatic endocarditis occurring in the practice of Dr. Cary B. Gamble, Jr., to whom I am indebted for the following notes:

Case of A. B.; white; aet. 24. Seen in March, 1899. The family history was negative. The patient had always been unusually healthy, with no history of past illness except that three years previously he had a chancre, for which he had taken a thorough course of treatment extending over three years. He was an amateur athlete and football player. When first seen he was suffering from obstruction of one side of the nose as the result of an old fracture of the septum, which caused a chronic post-nasal catarrh. The following note on the physical condition was made: Medium sized, powerfully built, athletic looking man. Lungs clear, expansion above the average. *Heart: apex in normal position, sounds clear, soft and strong.* The septum was straightened with relief of the nasal symptoms.

The following December, eight months later, the patient consulted Dr. Gamble as to the condition of his heart. Ten weeks before, while playing half-back on his football team, he was running through the opposing line with the ball, his body bent forward, when a player on the other side was seen to kick him violently in the chest. He fell, was picked up unconscious, and remained so for some time before he regained consciousness. A large bruised spot was seen over the cardiac area, but there is no mention of a fracture of the rib or sternum. He was ill in bed for four weeks with great weakness following. During this illness his symptoms were all cardiac; pain, shortness of breath, palpitation, and for the first few days fever of a moderate grade. The accident occurred early in October. December 20 the following note was made by Dr. Gamble: "General condition good. Pulse 90, regular, strong. Heart: apex in 5th interspace, ½ *inch outside nipple line*, visible, no thrill. At the apex there is heard a *loud, systolic murmur* which is transmitted back to the angle of the scapula and is heard all over the cardiac area with maximum intensity at the visible apex." A year after the above note was made, the condition of the heart was practically the same, except that the pulse rate had fallen to 70 and there was no consciousness of the heart's action. Dr. Gamble is of the opinion that the patient had a traumatic endocarditis involving the mitral valve, with resulting regurgitation.

The important points in this case are: (1) Eight months previous to the accident, the heart was entirely normal; (2) the contusion of the chest was not associated with fracture of the ribs or sternum; (3) the immediate onset of the acute cardiac symptoms with fever; (4) the development of chronic mitral insufficiency. On the other hand, the history of lues suggests the possibility of some pre-existing cardiac lesion, although the thoroughness of the treatment, the involvement of the mitral valve and the negative character of the previous heart examination are strongly against this.

In an examination of the comparatively few cases of traumatic endocarditis which have been reported they are found to fall into three groups:

1. Those in which symptoms of acute endocarditis develop, either at the time of the accident or within a short period afterwards, and which may or may not run into chronic endocarditis.

2. Cases in which at a varying interval after the trauma a chronic endocarditis gradually develops.

3. Cases in which following injuries in various parts of the body, without actual injury of the heart itself, micro-organisms gain entrance to the circulation and set up second-

ary inflammation of the endocardium. This group is only indirectly of traumatic origin and need not be considered, as the heart involvement is merely an accident in a more general infectious process.

Acute Traumatic Endocarditis.—The case just reported is an example of the first or acute type. In addition to this we have been able to find five other cases which fall into the same group. In these cases there is no question as to trauma being the immediate cause of the cardiac lesion. The heart, except in one case, is known to have been normal previous to the injury.

A brief synopsis of three cases in connection with the one just reported will give a fair idea of the similarity of the features which they present. The principal features of the remaining cases are given in the "Summary."

Litten's[7] *Case (II).*—A young German army officer had his chest crushed against a post by a horse. He had previously been examined several times by army surgeons and was known to be perfectly sound. Immediately after the accident he was seized with severe pain in the left chest. Within the next day or two typical symptoms of acute endocarditis *with fever* set in. In the second week a loud systolic murmur developed at the apex. In the fourth week the constitutional symptoms began to show gradual improvement. When seen by Litten, six months after injury, shortness of breath on exertion was still present, and examination of the heart showed enlargement of both ventricles, a systolic thrill and loud systolic murmur at the apex, an accentuated second pulmonic sound, and at the aortic area a very loud rough systolic murmur. Diagnosis: Mitral insufficiency and beginning aortic stenosis.

Litten also reports two other cases of acute traumatic endocarditis. These will not be considered here in detail, but the principal features which they present are shown in the "Summary," cases IV and V.

In *Luckinger's*[8] *case (III)* in a man 25 years old, following a contusion of the left chest without fracture of the ribs or sternum, palpitation of the heart, shortness of breath, cyanosis and fever developed on the third day. At the apex a double murmur was heard. There was also slight effusion into the pericardium. Complete recovery seems to have occurred within a few weeks.

The following case illustrates the anatomical condition in an acute inflammation of the endocardium a short time after injury.

In *Riedinger's*[9] *case*, there was an autopsy upon a woman 54 years old, three days after injury caused by a fall from a considerable height, without fracture of the ribs. Hæmorrhages were found under both the pericardium and endocardium with laceration of the mitral valve at the insertion of the papillary muscles. Some thickening of the papillary muscles indicated possible pre-existing disease. There was a *deposit of fibrin at the base of the mitral valve* at the point of laceration.

As previously stated, the special interest in these cases lies in the fact that the relation between trauma and the subsequent endocarditis is not questionable. The result of acute traumatic endocarditis as shown by these few cases may be recovery, chronic valvular disease, or death.

[7] Litten, Die Endocarditis und Ihre Beziehungen zu anderer Krankheiten. Verhand. Congress f. inner. Med., xviii, 1898.

[8] Riedinger, Ueber Herzaffectionen nach Unfällen. Monatschr. f. Unfallheilkunde, 1894, 351.

Chronic Traumatic Endocarditis.—Of the second group of cases, in which at a varying interval after the trauma, chronic endocarditis gradually develops, there are more numerous instances reported. Here endocarditis in nearly every case showed itself as chronic valvular disease. In this group we must be most careful in determining that trauma is the cause, and not a mere accidental occurrence in the course of endocarditis developing from other causes, especially if we have no note upon the heart previous to the injury. In selecting such cases I have carefully excluded all instances (a) in which there is reasonable doubt as to the previous condition of the heart, (b) where the interval between the injury and recognition of cardiac disease seemed too long, (c) where other etiological factors than trauma appear to have entered, or (d) where the cases are not reported in sufficient detail to determine the character of the heart lesion. In selecting only the cases which fulfill all these requirements, we probably exclude many cases which should be classed under traumatic endocarditis, especially those which develop after trauma in previously diseased hearts. In all some nine cases of chronic traumatic endocarditis have been found answering these requirements. Of these Litten[9] has reported 1; Allbutt,[10] 2; Riegel,[11] 1; Stern,[12] 1; Heidenhain,[13] 1; Riedinger,[14] 1; Oppenheim,[15] 1; Ritter,[16] 1. I have not thought it advisable to refer in detail to these cases individually, but to present a brief analysis of them together with the five acute cases which later developed into chronic valvular disease; in all 14 cases. Such an analysis brings out some rather interesting points. The appended "Summary" shows the principal features of each case in a tabular form.

Location of the contusion.—Of these 14 cases in 12 instances the contusion seems to have been over the præcordia or about the anterior part of the chest; in 2 cases (Stern and Oppenheim) there was a fall from a ladder upon the back (*contre-coup*).

The extent of the external injuries.—The severity of the external injury varies. While in one case this was sufficient to fracture the sternum, in no case was there a fracture of the ribs reported. In several of the cases there seems to have been no external evidences whatever of injury.

[9] Litten, Ueber traumatische Endocarditis. Aerztliche sachverständige Zeitung, 1900, No. 24.

[10] Allbutt, On Overwork and Strain of the Heart and Aorta. Trans. Clin. Soc., Lond., vi, p. 112.

[11] Riegel, Zur Lehre von der chronischen Myocarditis. Zeitschrift f. klin. Medizin, xiv, p. 328.

[12] Stern, Ueber traumatische Entstehung innerer Krankheiten. Jena, 1900, p. 21.

[13] Heidenhain, Ueber die Entstehung von organischen Herzfehlern durch Quetschung des Herzens. Deutsche Zeitschrift für Chirurgie, Bd, xli, 1895.

[14] Riedinger, Ueber Herzaffectionen nach Unfällen. Monatsch. f. Unfallheilk., 1894, 351.

[15] Oppenheim, Die traumatischen Neurosen. Berlin, 1892, Beob., xxv, p. 69.

[16] Ritter, Mitralstenose in Folge Fractura Sterni. Berl. klin. Wochenschr., 1889, p. 699.

Subsequent valvular disease.—In every instance the result was chronic valvular disease, except in one case (III), where the murmurs ultimately disappeared.

Character of the valvular lesion.—Of the type of the valvular lesion we find in 6 cases mitral stenosis alone; in 3 cases mitral insufficiency alone; in 1 case aortic stenosis; in 1 case mitral insufficiency and aortic stenosis (aortic endocarditis); in 1 case mitral insufficiency with stenosis and aortic insufficiency with stenosis; in 1 case mitral endocarditis; in 1 case aortic insufficiency. *The interval after the injury* when valvular disease was established cannot be accurately determined. The only data we can usually give is the time of the first recognition of the murmur. In 2 cases this was heard soon after the injury, on the 10th and 14th days, respectively; in 1 case 18 days; in 2 cases 6 weeks; in 1 case 4 months; in 1 case 5 months; in 2 cases 8 months; in 1 case 12 months; in 2 cases 18 months.

Symptoms.—In about half the cases, following the injury there was *pain about the heart.* In 7 cases the blow on the chest is stated to have produced *unconsciousness.* In 6 cases distressing cardiac symptoms came on immediately after the injury, while in 4 cases acute symptoms with fever developed within a few days. In the remaining cases there were only indefinite symptoms of heart injury until several weeks or months afterwards. In 5 cases *fever* is mentioned as occurring in connection with other acute symptoms of endocarditis. In only 1 case is the chronic valvular disease reported as terminating fatally. It seems unnecessary to continue this analysis further, as in other respects valvular disease resulting from traumatic endocarditis appears to resemble chronic valvular disease of non-traumatic origin.

To sum up the facts just presented, we can say that following an injury about the thorax, endocarditis may develop rapidly, or come on gradually after the elapse of a considerable interval. Chronic valvular disease is the usual outcome.

Unconsciousness following the injury in some cases may be the result of extreme shock to the nervous mechanism of the heart directly or reflexly. The cardiac involvement may be out of all proportion to the extent of the external injuries. The injury while usually direct, appears at times to be by *contre-coup.* The mitral valve is affected in a very large proportion of cases. The frequency of mitral stenosis is to be noted. Injuries of the right heart are comparatively rare. The onset of acute cardiac symptoms with fever, a short time after the injury, which occurs in some of the cases, points to a secondary infection of the injured endocardium, as also does the progressive character of the lesion in the more chronic cases. In general the subsequent history of the cases is similar to other forms of valvular disease.

Many of the facts which have just been pointed out are explained when we study the effects of external trauma upon the endocardium and myocardium as seen at autopsy after death from severe thoracic injuries (Riebold[16]). The interior of the heart may merely show scattered patches of ecchymosis beneath the endocardium. There may or may not be actual laceration of the endocardium. Hemorrhage often extends into the myocardium which is bruised or lacerated. Actual rupture of the heart has been found. There is often laceration of the papillary muscles and chordæ tendineæ, especially of the mitral valve. This explains the frequency of subsequent mitral disease. Actual complete rupture of a valve curtain seems rare, but does occur. All this may occur in a perfectly healthy heart, although the susceptibility of a previously diseased heart would seem to be especially great. The period in the cardiac cycle at which the contusion occurs, may possibly influence the character of the lesion. A blow upon the ventricle when filled would seem to throw an unusual strain upon the papillary muscles and chordæ tendineæ of the mitral and tricuspid valves, while an empty ventricle would favor a contusion of the endocardium or myocardium.

It does not seem well to pass over this subject without referring to "*rupture of the heart valves,*" which is known to occur occasionally, although in this connection we are concerned more with the remote effects of trauma, than with the direct mechanical effect of actual rupture of the valves. In actual rupture symptoms of insufficiency with cardiac murmurs develop immediately after the laceration of a valve segment. Stern has analyzed 35 cases collected by Barié.[17] Of this number 23 were classed by Barié as "spontaneous;" i. e. they occurred in a diseased heart, either without apparent cause, or as the result of an increase in the arterial tension produced by a sudden muscular exertion. The remaining 12 cases were considered by Barié as "traumatic" in origin. Of these 12 there were 9 instances of rupture of the aortic valve and 3 instances of rupture of the mitral. Stern has made a careful analysis of the cases and comes to the conclusion that in all of the 9 aortic cases it is impossible to exclude a sudden increase in the arterial tension produced by exertion acting upon a diseased valve, as the immediate cause. However, I am unable to see why trauma might not produce rupture of an aortic segment by increase in the aortic blood pressure through a contusion of the large vessels of the thorax. Of the 3 "traumatic" cases of ruptured mitral valve, 2 which came to autopsy did not show rupture of the valve itself, but merely of a few papillary muscles. Stern concludes that actual rupture of an aortic valve from trauma alone is very rare, and that in the so-called "traumatic ruptures" of the mitral valve we are usually dealing with an endocarditis developing from an injury of the endothelium or papillary muscles, rather than with actual rupture of a valve segment itself.

Nature of the inflammation.—Autopsy reports unfortunately throw but little light upon the character of the reaction in the endocardium following trauma. It would seem possible that in some cases the inflammation is of a simple, non-infectious character; i. e., there may be merely the formation of granulation tissue at the injured point, without a secondary invasion of micro-organisms. If such scar tissue is in a posi-

[17] Barié, Recherches clinique et expérimentales sur les ruptures valvulaires du coeur. Rev. de Médicine, 1881.

tion to project into the blood current, a murmur might even develop, but it is hard to understand how such a simple non-infectious process could be progressive in character, unless it was very extensive, or so placed as to actually interfere with the function of the heart or valves.

In cases of traumatic endocarditis going on to chronic valvular disease it seems certain that there has been a secondary infection of the injured part by micro-organisms which have gained entrance to the circulation. The presence of fever and other symptoms of an acute infectious endocarditis, as well as the progressive character of the lesions in the more chronic cases, as illustrated by the frequent occurrence of mitral stenosis, can scarcely be explained on any other grounds. Just how bacteria gain entrance to the circulation

SUMMARY OF CASES OF ACUTE AND CHRONIC TRAUMATIC ENDOCARDITIS DEVELOPING INTO VALVULAR DISEASE.

Reference	Sex: Age	Character and Location of External Injury	Fracture of ribs or sternum	Symptoms at time of injury	Onset of Cardiac Symptoms	Cardiac Symptoms	Fever	First recognition of valvular disease	Valvular Lesion	Complications	Result
I. Case here reported	Male: 24	Kick on chest	None	Unconsciousness	First or second day	Dyspnœa, palpitation; pain about heart	Present first few days	6 weeks	Mitral insufficiency	....	Condition practically unchanged 1 year later
II. Litten[9]—Case 2	Male:	Chest crushed	None	Pain; not unconscious	Immediate	Dyspnœa; rapid pulse; pain about heart	Present first few days	2 weeks	Mitral insufficiency and aortic stenosis	....	Marked symptoms 6 months later
III. Luckinger[3]	Male; 25	Contusion of left chest	None	Slight	3d day	Symptoms of acute endocarditis	3d day	Uncertain	Mitral insufficiency	Pericarditis with effusion; traumatic pleurisy	Disappearance of murmur in few weeks
IV. Litten[9]—Case 3	Male: 32	Severe blow on left chest with handle of windlass	None	Unconsciousness?	Immediate	Pain over heart; shortness of breath; palpitation; rapid pulse; irregularity	....	18 days	Mitral endocarditis	....	Uncertain
V. Litten[9]—Case 4	Male; 21	Contusion on chest and back	None	Unconsciousness	Immediate	Pain about heart; shortness of breath; palpitation; irregularity	Present soon after injury	10-12 days	Mitral insufficiency	Onset of dry pericarditis 8 days after injury—lasted a few days—completely cleared up	Chronic mitral insufficiency
VI. Litten[9]—Case 1	Male; 47	Severe fall on left chest	None	Unconsciousness	Very gradually a few days later	Rapid pulse; palpitation	None mentioned	8 months	Aortic insufficiency	....	Present 3 years later
VII. Allbutt[10]—Case 1	Male; 14	Kick on chest	None	Oppression about the heart; pain and palpitation	Immediate	"All the usual symptoms of mitral stenosis"	....	18 months	Mitral stenosis	....	Subsequent history unknown
VIII. Allbutt[10]—Case 2	Male; 16	Blow on chest with cricket ball	....	Unconsciousness	Immediate	Palpitation	...	12 months	Mitral stenosis	....	Unchanged 12 months later
IX. Riegel[11]	Male; 44	Kick on chest	None	Pain, shortness of breath	Gradually developed in 4 weeks	Œdema; cyanosis; irregularit ; shortness of breath	None mentioned	18 months	Mitral stenosis	Myocarditis	Death 4 years later
X. Stern[12]	Male; 35	Fall on back from ladder	None	Principally referable to other injuries	Gradually after several days	Pain about heart	None mentioned	6 weeks	Aortic stenosis	....	More marked in 3 months
XI. Heidenhain[13]	Male; 49	Severe contusion of chest	None	Pain and shortness breath	Gradual development	Cardiac pain; palpitation; shortness of breath	None mentioned	5 months	Aortic insufficiency; aortic stenosis; mitral insufficiency; mitral stenosis	....	Living 5 months later
XII. Riedinger[14]	Male;	Contusion of chest	None	None	3d week	Apparently those of acute endocarditis	3d week	Uncertain	Mitral stenosis	....	Uncertain
XIII. Oppenheim[15]	Female; 23	Fall on back from ladder	None	Unconsciousness	3d week	Palpitation and very rapid pulse	....	8 months	Mitral stenosis	....	Unchanged
XIV. Ritter[16]	Male; 34	Severe blow on chest	Sternum fractured	Unconsciousness	Immediate	Palpitation; severe cardiac pain	None	4 months	Mitral stenosis	....	Symptoms marked 4 months after injury

cannot be certainly determined. Litten[18] thinks that a slight associated traumatic focus in the skin or lungs in these cases, may be the portal of entry. In experiments upon animals in which the endocardium is injured by passing a probe into the interior of the heart through the great vessels, unless secondary infection occurs, healing is rapid and complete. While it is doubtless true that the endocardium is often injured and healing takes place as in wounds elsewhere without producing permanent alterations in the heart, it is important to bear in mind that such injuries, as in the cases here brought to your attention, may be the starting point of subsequent progressive valvular disease.

Traumatic Myocarditis.

In discussing traumatic pericarditis and endocarditis, reference has already been made to the effect of trauma upon the heart muscle, and we have seen that, following external contusion of the thorax, hemorrhages into the muscle, contusion, laceration, or even rupture of the heart wall may occur. Moreover, in traumatic inflammations starting in the pericardium or endocardium some reaction in the adjacent muscle may follow, giving rise to more or less inflammation of the myocardium. The muscle fibers destroyed by trauma are replaced in the repair process almost entirely by connective tissue. Unless the extent of the injury is great, or there is subsequent infection of the contused area, the development of progressive myocardial disease seems unlikely in the case of a previously sound heart. Yet this sometimes does occur. If, however, we are dealing with a heart already the seat of myocardial disease, or one in which the reserve resistance is lowered by pre-existing hypertrophy, the liability to serious myocardial changes following injury is increased. That so few cases of traumatic myocarditis are reported is probably to be explained in the following way: (a) In severe laceration of the heart muscle, death usually results. (b) In injuries of moderate severity in a normal heart, repair without ill effects probably occurs in the majority of cases, while if myocardial changes do take place, they are in association with serious endocardial or pericardial disease and are obscured for a time by them. (c) If, however, we are dealing with a previously diseased heart, even if the relation between trauma and myocarditis seem close, we feel that we should be cautious in necessarily assuming a causal relation.

One of the cases here reported under traumatic pericarditis is also an example of infarction of the myocardium developing in a previously diseased heart as the result of trauma. We are fortunate in this case in having the causal relation between trauma and the subsequent myocardial lesion established by the histological findings, which show a process of about the same duration as the injury.

The clinical features of the case have already been referred to in sufficient detail. It may be recalled (Pericarditis, Case II) that the patient was known to have had an enlarged heart a year and a half before injury, but had not previously shown acute symptoms of a diseased myocardium. Immediately following the injury, gallop-rhythm, rapid pulse and other signs of defective heart action were noted. Autopsy four weeks after injury showed a recent infarct of the myocardium in addition to the fresh adherent pericardium already referred to. This infarct was about 1 cm. in diameter, and was situated in the wall of the left ventricle. Macroscopically it was grayish in color and of the usual wedge shape. On microscopical examination it was found to be made up almost entirely of muscle fibers beginning to undergo necrotic changes. The muscle fibers are cloudy, have lost their well-defined outlines, and the nuclei show fragmentation. Scattered among the disintegrating muscle fibers are numbers of fibroblasts and embryonic connective tissue cells. The whole picture is that of a recent necrosis of the myocardium with commencing connective-tissue formation. The process appears to be of about the same age as that already described in the pericardium, and to date from about the time of the injury.

Just how trauma produced infarction of the myocardium is not certain, but injury of one of the branches of the coronary artery by contusion of the heart wall, would explain the lesion. At all events there seems to be some close association between the injury and the formation of the infarct. The myocardium showed no other changes except a general hypertrophy of the walls of both ventricles.

We have been able to find four cases of myocarditis reported in which the causal relation between trauma and myocardial disease seems established. The condition seems of sufficient rarity to justify a brief synopsis of these cases.

1. *Mendelsohn's*[18] *Case.*—Strong and healthy man, 26 years of age. Previous examinations of heart negative. He was first examined three weeks after his chest had been crushed against a wall by a vicious horse. He was rendered breathless by the injury, but did not become unconscious. There was considerable pain about the chest, with weakness and shortness of breath. On examination, three weeks after the injury, he was found to be short of breath and cyanotic. Pulse small, rapid and irregular. Heart distinctly enlarged, especially to the right. There were no murmurs. His condition gradually grew worse, and œdema developed which yielded to digitalis. Eight months after the injury his condition is reported as serious, with marked symptoms of myocarditis.

2. *Stern's*[19] *Case (I).*—Man 19 years old, sustained a severe contusion of the chest without evidence of external injury. Examination of the heart after the injury absolutely normal. Two days after the accident traumatic pneumonia and pleurisy developed. With the clearing up of the pulmonary condition rapid pulse and cyanosis persisted, but there were no murmurs, and the heart dulness was not increased. For several months attempts to work were followed by shortness of breath. An examination eleven months after injury showed increased heart dulness, persistent small and weak pulse but no heart murmur. Three years later typical signs of myocarditis were present. Marked cyanosis, dyspnœa, small irregular pulse and pulsation of vessels of neck. There was great increase in the heart dulness,

[18] Mendelsohn, Ein Fall von traumatischer Myocarditis. Deutsch. med. Wchnschr., 1898 (xxiv), Ver. Beil., 25.

[19] Stern, Über traumatische Entstehung innerer Krankheiten. Jena 1900, p. 46.

especially to the right and at the apex and tricuspid area, a soft systolic murmur.

3. *Stern's*[20] *Case (II).*—This second case is somewhat similar to the last, but symptoms of myocarditis did not develop as rapidly after the injury, so that a causal relation is not as certain as in the last case. As in the last case, the signs of myocarditis were typical. There were also associated nervous disturbances in the heart's action.

4. *Riegel's*[21] *Case.*—Here there seems to be little doubt of the relation between a severe prækordial contusion and cardiac symptoms which set in immediately afterwards, in the case of a man 44 wears old who was kicked by a horse. The condition gradually became worse, symptoms of myocarditis and mitral stenosis developing, and resulting in death four years later. At autopsy, in addition to mitral stenosis, there was *extensive fibro-myocarditis of the left ventricle*, especially in the anterior wall near the apex.

5 and 6. *Hochhaus's*[22] *Cases.*—Two cases are cited by him. In both of these cases the relation between trauma and myocarditis, while probable, cannot be said to be definitely established. Stern refers to other reported cases of so-called traumatic myocarditis, but the part played by trauma seems open to question.

From an examination of the foregoing cases, and a study of autopsy reports, it can scarcely be doubted that myocarditis occasionally develops as the result of contusions of the chest. From the scarcity of reported cases, myocarditis of traumatic origin cannot be common, although the traumatic origin of some cases is probably overlooked. As in other traumatic affections of the heart, the evidences of external injury probably bear but little relation to the severity of the cardiac injury. Myocarditis may thus develop in a heart previously sound, but the susceptibility of diseased heart muscle must certainly be greater. Disturbances in the nutrition of the heart, by interference with the blood supply, or by actual destruction of the muscle substance, are probably the usual form of injury. In the case here reported there was an infarct of the myocardium. The left ventricle is probably the one most frequently injured. Symptoms may develop immediately, or come on gradually after injury. Traumatic endocarditis may be associated with traumatic inflammation of the pericardium and endocardium. That serious myocarditis can develop as the result of the direct extension of traumatic pericarditis, as asserted by Bamburger[23] and Friedreich,[24] seems quite probable. In other respects myocarditis of traumatic origin does not seem to differ from the non-traumatic forms.

Other Effects of Trauma Upon the Heart.

"Dislocation of the heart."—This is claimed to have occurred as the result of trauma in a case reported many years ago by Stokes.[25] It seems probable, however, that the displacement of the heart to the right was secondary, and the result of adhesions to the right pleura and shrinkage of the right lung, rather than actual mechanical displacement at the time of injury.

Aneurysm of the heart.—The older writers referred to the development of the aneurysm of the heart wall from trauma. Cases are said to have been reported by Dionis and Zannine. The reports are so meagre that definite conclusions cannot be drawn from them. No example of aneurysm of the heart following contusion of the chest has been found in the recent literature. A case is reported by Mühlig[26] in which aneurysm of the right ventricle followed a stab wound of the heart. There seems to be no reason why aneurysm should not develop in a heart weakened by trauma or by subsequent connective tissue formation, but with this exception no case of undoubted traumatic origin has been found.

Cardiac neuroses.—After accidents marked disturbance in the action of the heart may develop. This can occur after concussion in which there may or may not be any actual thoracic injury, or it may follow mere mental shock. Either immediately after the injury or at a varying interval, associated with other symptoms of hysteria or neurasthenia, there may be a great increase in the rapidity of the heart's action, or even some irregularity. This condition must be carefully differentiated from traumatic myocarditis. The condition is probably a purely functional one, as in the case of other traumatic neuroses, and is not due to any special injury of the nervous mechanism of the heart.

It should also be borne in mind that pre-existing disease of the heart, whatever its nature, may be aggravated as the result of accidents of various kinds. This may be brought about in several ways; as the direct effect of actual concussion of some part of the body, as the result of some unusual muscular exertion at the time of the accident, or reflexly through the psychical influence of the accident. All three of these factors must be considered in the exacerbation of cardiac disease following accidents.

From a study of the cases here presented, it seems established that not only may pre-existing disease of the heart be aggravated by trauma, but that in a heart previously sound, pericarditis, endocarditis or myocarditis may develop as the result of cardiac injury produced by contusions about the thorax, often without any external evidence of trauma.

Discussion.

DR. MACCALLUM.—I have seen in one of these cases the sections from the pericardium, but I am not convinced that they represent a simple, non-bacterial form of inflammation. There was a great deal of exudate and a good deal of proliferation of granulation going on, and it is difficult to understand how that could take place without the intervention of bacteria or some other irritating material. As to the occurrence of *contre-coup*, that seems difficult to understand also in view of the fact that the structures in the chest are so elastic. The

[20] Idem, p. 50.

[21] Riegel, loc. cit.

[22] Hochhaus, Beiträge zur Pathologie des Herzens. Deutsche Uebersetzung von Becker, Leipzig, 1836, 171.

[23] Bamburger, Lehrbuch der Herzkrankheiten. Vienna, 1857, 160.

[24] Friedreich, Krankheiten des Herzens. Virchow's Handbuch der spec. Pathol. u. Therap., v, 2, Erlangen, 1861, p. 153 et seq.

[25] Stokes, A case of probable dislocation of the heart from external violence. London Med. Gaz., 1831, viii, 560

[26] Mühlig, Constantinople. Gaz. Med. d'Orient, iv, Sep. 6, 1860.

contre-coup with which we are familiar takes place most often in the skull, where the contents are enclosed compactly in a rigid structure, a condition entirely different from that in the chest.

Dr. McCrae.—I was very much interested in this paper and I think a point of special importance is in reference to the probability of the association of bacterial infection. It would seem reasonable to suppose that one could have such a condition with or without organisms, just for instance as a simple inflammation in the knee joint may occur without bacteria. Like Dr. MacCallum, I am inclined to doubt the possibility of its production by *contre-coup,* not only for the reasons he gave, but because Dr. Pleasants said that the right heart nearly always escaped.

Dr. Pleasants.—I think myself that there is usually a bacterial infection, but I do not see why we may not have a murmur, for instance, where the endocardium about the valves has been injured without infection taking place, if the resulting scar tissue so projects into the blood current as to interfere with the flow. Unless subsequent infection occurs such a lesion would probably not be progressive.

In the case of the pericardium I see no reason why at times we may not have a single non-infectious inflammation such as is known to develop in the synovial membrane of joints after trauma.

In regard to the possibility of injury of the heart and its membranes by *contre-coup,* I see no anatomical objections to its occurrence. It seems the most reasonable explanation of single localized pericarditis following blows on the back. Dr. McCrae's objection to *contre-coup* as a possible course of endocardial injury, because of the rarity of traumatic valvular disease of the right side of the heart, is hardly satisfactory when we consider that even after severe blows directly over the præcordia, the left and not the right side of the heart, is the one affected in the great majority of cases of traumatic endocarditis. I think that the true explanation of the relative frequency of chronic valvular diseases of traumatic origin on the left side is to be found not so much in the actual escape from injury of the right heart, but in the greater liability of the injured endocardium on the left side to become the seat of subsequent progressive degenerative changes. The greater susceptibility of the left heart to non-traumatic endocarditis is too well known to require comment.

GIANT CELL SARCOMA OF BONE.[1]

REPORT OF A CASE OF A MEDULLARY GIANT CELL SARCOMA OF THE UPPER END OF THE TIBIA IN WHICH THE TUMOR WAS APPARENTLY COMPLETELY REMOVED BY CHISELLING WITHOUT DESTROYING THE CONTINUITY OF THE TIBIA, AND A DISCUSSION OF THE FACTS WHICH JUSTIFY THIS MORE CONSERVATIVE PROCEDURE.

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Path. No. 4530 (Private Case).—Giant cell medullary sarcoma, occupying the upper end of the tibia. Trauma (contusion) nine years, local pain and tenderness since, symmetrical expansion of the upper end of the tibia two and one-half years. Operation December 4, 1902, curetting and chiselling. Result, April 12, 1903, apparently well.

Clinical History.—White, male, aged 29 years. Nine years ago there was a distinct trauma, a contusion of the upper end of the tibia. At the time of the injury the patient felt only a slight discomfort. He was able to get up and walk about. There was no joint effusion and no locking of the joint. The upper end of the tibia was quite painful and tender for three or four days. Since this injury the patient has always been conscious that the upper end of the tibia was very sensitive to slight contusions. He noticed that when he struck this bone the injury, no matter how slight, caused more pain than if he struck any other bone. Pain without trauma was not at first a symptom, but tenderness has always ben present since a short time after the injury, and on the whole the tenderness to slight contusions has increased. Within the last few years he has noticed that if he walked a long distance his attention would be called to the upper end of the tibia by dull pain. It is a very important symptom that since this slight contusion this part of the bone has been unusually tender, a condition not present in normal bone; also after exertion and traumatism it is painful.

He has observed swelling for at least two and a half years. Without much doubt the swelling has been present longer. The swelling has been very gradual, and even now it is but slight. He does not think that the swelling has been any more rapid recently. The tenderness has been more marked within the last year, and the pain after exertion has also increased within this time. This history almost excludes osteomyelitis. The diagnosis lies between a giant cell medullary sarcoma and a bone cyst.

Examination.—On inspection there is a uniform and symmetrical expansion or enlargement of the upper end of the tibia. It is most marked on the medial and lateral surfaces which are not covered by muscle. The normal concavity of the bone at this place is occupied by a convex

[1] Reported before the Johns Hopkins Medical Society, February 15, 1903, with exhibition of patient.

swelling. On palpation the upper end of the tibia, the so-called head, is found expanded. The surface of the bone feels rougher than the normal smooth shaft beneath. This roughness is composed of fine roundish elevations like a mulberry. It feels very hard, it is not compressible; palpation elicits quite marked tenderness. There is no pulsation.

I have never observed a cyst of a long bone but from the cases in the literature the tenderness in this case seemed more marked than that observed in a benign bone cyst.

X-ray Examination (see Figs. 1 and 2).—Fig. 1 is the antero-posterior view. The shadow in the upper end of the tibia is quite characteristic of a medullary soft giant cell sarcoma when compared with other Roentgen pictures in which the diagnosis has been confirmed by operation. The characteristic features of this shadow are the fine darker

FIG. 3.

lines dividing the light area into lobules of various sizes. The line of demarkation between the light area and the shaft beneath is not very sharp. The dark zone about the light area indicates that the expanded shell of bone is thin. There is no evidence that the tumor has infiltrated into the joint. Fig. 2, a lateral view, is not as good a photograph: it is apparently a little overexposed. This photograph, however, shows the irregular formation of new bone beneath the periosteum on the anterior surface of the tibia.

Operation, December 4th, 1902.—An exploratory incision was first made without the Esmarch bandage. The knife demonstrated that the shell of bone on the medial anterior surface was very thin, and the incision exposed a very hemorrhagic, reddish, soft, friable tumor, a typical giant cell sarcoma. The Esmarch was then applied to the thigh. The upper end of the tibia was first exposed by dissecting flaps of skin. The shaft below the expanded shell was exposed; here the periosteum and the bone were normal. The shell of bone which was expanded above the normal shaft differed not only in its expansion, but in its character. The periosteum could not be separated from it, and the bone was rough. With the chisel a piece of the normal shaft and the shell of bone occupying the anterior medial surface of the head of the tibia were removed. [The shell of bone varied from 2 to 3 mm. in thickness.] This exposed a soft, very friable and hemorrhagic mass, completely filling the medullary cavity. The tumor had uniformly expanded the upper end of the tibia. Almost the entire tumor was shelled out, leaving a large bone cavity about the size of a man's fist. The tumor removed is shown in Fig. 3. It could be easily separated from the medullary tissue of the normal shaft below. The shell of bone left appeared to be from 4 to 7 mm. in thickness. It was not a perfectly smooth surface, but had small cavities (recesses), containing tumor tissue. These cavities were curetted. The bone appeared denser than the normal shaft, but in addition to the cavities mentioned containing tumor tissue, there were pin-point and line depressions; the appearance of bone undergoing absorption. The tumor at no point penetrated the shell of bone, nor did it extend to the articular cartilage of the knee-joint. The entire bone cavity was curetted and swabbed with pure carbolic acid, followed by alcohol, then irrigated with 1:1000 bichloride solution followed by normal salt solution. After this thorough curetting and cleansing the surface of the shell of bone showed to the naked eye nothing suggesting tumor tissue. The wound was then tightly packed with sponges and the Esmarch removed. After waiting a few minutes the sponges were removed from the cavity one by one, and it was found that the shell of bone was perforated by at least twenty-five or thirty quite large vessels, at least as large as the temporal artery. It was necessary to plug each one of these openings with Horsely wax. The oozing from the bone between the vessels mentioned was but slight. This use of the Horsely wax completely checked all bleeding. The bone cavity left at the operation measures at least 7 cm. in length, its greatest width just beneath the knee-joint 4 cm.

Microscopic Study.—The tumor is pretty uniform in its histological picture. It is composed of large giant cells, the nuclei of which are centrally arranged. The stroma of the tumor between the giant cells is a rather loose connective tissue, containing small round and some spindle cells. This connective tissue is very vascular. The histological picture corresponds to the common giant cell epulis.

Post-Operative Notes.—There was no hemorrhage. It was at least two weeks before all the wax was removed. On removing the wax the bone surface was covered with fine red granulation tissue, and with the point of a knife it could be easily demonstrated that new bone was present in this granulation tissue. A microscopic study was made of two pieces of this tissue. Histologically it corresponded to the usual picture of vascular granulation tissue. Here and there one saw a few giant cells which, however, were smaller and contained fewer nuclei than the giant cells of the tumor.

March 1, 1903.—Three months since operation. The

patient has been walking without crutches since the sixth week. There is no restriction of motion at the knee-joint. The cavity is rapidly filling up. The lining granulation tissue is firm and finely granular. At no point is it œdematous or exuberant. It is infiltrated with new bone tissue. The naked eye appearance is that of normal bone granulation tissue. We would expect, if there was a recurrence of the tumor, that the tissue lining the bone cavity would form more rapidly, and assume the appearance of œdematous, friable, tumor tissue, which we have observed in other cases. An anterior and lateral X-ray (Fig. 4 and 5) photograph taken February 28 demonstrates that the shadow of the bone cavity is smaller than the shadow of the original tumor, and the present shell of bone about the cavity is considerably thicker than that about the tumor. There is therefore no evidence yet of a recurrence of the tumor.

Remarks.—I felt justified in attempting to cure this case by curetting rather than amputation because of the following facts brought out in a recent study of bone sarcoma. Among 42 cases of sarcoma of bone observed in Prof. Halsted's clinic in the Johns Hopkins Hospital, we have observed 7 cases of giant cell sarcoma; 3 periosteal; and 4 medullary. These cases may be summarized as follows:

Surg. No. 2706.—Periosteal giant cell sarcoma, surrounding the shaft of the lower third of the ulna. Colored female, aged 45, tumor one year, pain nine months, the tumor pulsates. The patient refused amputation. The operation by Dr. Halsted consisted of resection of both bones of the forearm. The microscope demonstrates a pure giant cell sarcoma. The giant cells infiltrate the muscles slightly beyond the capsule of the tumor. The shaft of the ulna is slightly eroded. The stroma between the giant cells is a very vascular loose connective tissue.

Result.—March, 1903, nine years and six months after the operation, well. The woman uses the arm and supports herself by washing and ironing.

Surg. No. 5696.—Periosteal giant cell sarcoma situated on the anterior surface of the shaft of the tibia at the junction of the upper and middle third. White, male, aged 35. Localized pain in the shaft of the tibia fourteen months. Tumor observed twelve months. The tumor when first observed was the size of a pea and apparently adherent to the periosteum or bone. At the end of eight months it had grown to the size of a hickory nut. It was excised at this time and recurred immediately in the scar. We were not informed of the extent of this operation. The recurrent tumor is about 9 by 3 cm. in diameter. It was adherent to the bone but not to the skin. The operation consisted of excision of the tumor together with an area of healthy tissue and a piece of apparently not infiltrated shaft of the tibia.

Gross Pathology.—This tumor was encapsulated and situated apparently between the periosteum and the shaft of the bone. It was composed of soft, vascular and friable tissue, and contained numerous small cavities filled with blood. The bone was not infiltrated.

Result.—March, 1903, six and a half years after the operation, well.

Surg. No. 12,235—Periosteal giant cell sarcoma situated over the head of the tibia. Female, white, aged 48. The patient noticed a tender area over the left tibia two years ago. A tumor was not observed in this area of tenderness until one year ago. In four months it had grown to the size of a walnut. At this time it was excised by her physician. Recurrence took place at once. A few weeks ago the skin over the tumor ulcerated and a fungous mass protruded. The fungous mass measured about 3 by 3 cm. in diameter and projected 1 cm. above the surrounding skin.

Operation.—Amputation of the thigh through the condyles of the femur.

Gross Pathology.—The fungous tumor is circumscribed. It originates from the periosteum. The outer table of the tibia beneath is but slightly eroded. The center of the tumor is necrotic. Surrounding the necrotic area the tumor tissue is firm, not very friable, but slightly hemorrhagic.

Microscopic Sections.—The tumor is composed of many giant cells. The matrix of the tumor is composed chiefly of spindle cells. Many of the spindle cells are long and some are fibers. The bone is not infiltrated by the tumor cells. The pathological study in this case demonstrates that the tumor could have been completely removed by resection without injuring the continuity of the tibia.

Result.—March, 1903, one year and six months after operation, well.

Remarks.—The first two cases correspond to the so-called pure giant cell sarcoma epulis springing from the periosteum of the alveolar border of the upper or lower jaw. We have observed about ten cases; none have recurred after excision without destroying the continuity of the jaw. The third case corresponds to the fibro-spindle-cell-fibroma epulis of the jaw, except it contains more giant cells. We have observed about six cases; none have recurred after excision.

The giant cell periosteal sarcoma of the long pipe bones is not a very common tumor. It is without much doubt one of the least malignant forms of bone sarcoma, and in the majority of instances should be treated by excision without destroying the continuity of the bone.

Surg. No. 313.—Medullary sarcoma occupying the condyles of the femur. This case is clinically of interest because the symptoms of the new growth simulated a chronic arthritis. The patient was a white male 22 years of age. For seven months he suffered with pain and weakness referred to the left knee-joint, but continued to walk and work. Swelling of the knee had been observed only one month. The patient sought surgical advice because of the great increase of pain and swelling in the knee during the last three days. There was no history of gonorrhœa or lues. The clinical picture was one of effusion into the knee-joint. At the exploratory arthrotomy the synovial membrane was slightly injected and the joint contained clear fluid. It is not noted in the history whether there was any enlargement of the lower end of the femur. The case was observed before the advent of the

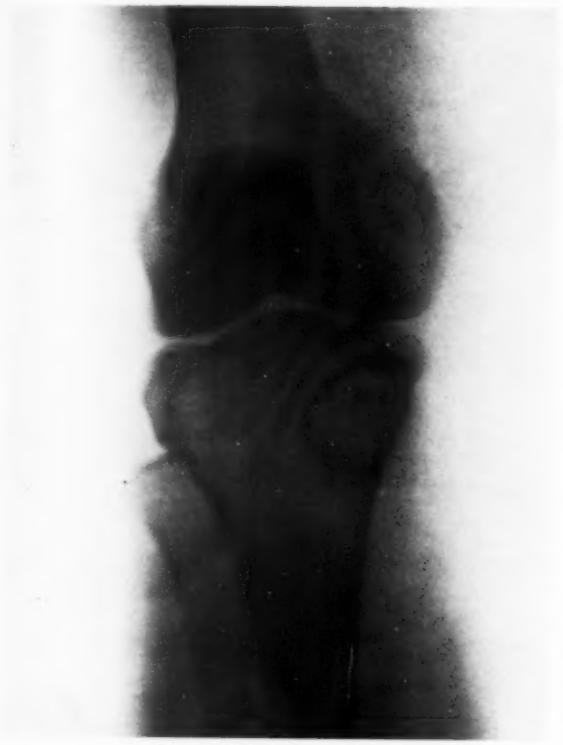

FIG. 1.

FIG. 2.

FIG. 4.

FIG. 5.

X-ray. The patient was observed from February 7 to April 16. The arthrotomy gave no relief. Pain continued and the effusion recurred. The aspiration fluid in March contained blood corpuscles.

Second Operation, April 16, 1890.—At the exploratory arthrotomy the effusion was very hemorrhagic. The joint contained a soft, friable, gelatinous, hemorrhagic tissue which perforated the external condyle of the femur and was continuous with similar tumor tissue in the external condyle. The thigh was amputated at the junction of the middle and lower third ten days later. The delay in the amputation was due to the necessity of getting the patient's consent. This patient died eight months after the amputation with symptoms of acute nephritis. There was no positive evidence of internal metastasis.

The microscopic study made at that time states that the tumor was composed of giant and very large spindle cells. For this reason it is a question whether this tumor may be considered a pure giant cell sarcoma.

The remaining three cases of medullary sarcoma, all of which I saw clinically and examined pathologically, are examples of the pure giant cell variety. All of these patients have remained well since operation. In three cases the tumor occupied the lower end of the radius, the fourth the upper end of the tibia.

Surg. No. 3815.—Medullary sarcoma lower end of radius. White male, aged 26 years. The pain and swelling of the lower end of the radius is of three months' duration following traumatism. At the exploratory incision into the pulsating tumor it was found that the lower end of the radius was expanded and occupied by a very vascular, soft, friable tissue, much disintegrated by hemorrhage. A thin shell of bone was preserved everywhere except on the extensor surface. In this area the tumor tissue was covered by thickened periosteum. The arm was amputated below the elbow. The study of the pathological specimen demonstrated that the tumor was completely circumscribed by periosteum and a shell of bone. The probabilities are that it could have been completely removed by resection of about 5 cm. of the lower end of the radius. Microscopically it was composed chiefly of giant cells. The tumor was almost completely disintegrated by hemorrhage.

Result.—March, 1903, 7 years and 4 months, the patient remains well.

Surg. No. 6532.—Medullary sarcoma of the lower end of the radius. This case is of particular interest on account of its relation to Colles' fracture. It is our only observation in which a tumor formed at the site of a fracture. The patient was a white male 45 years of age. Two years and four months ago a Colles' fracture took place after a fall on the palm of the hand. It is to be noted, however, that four months prior to this injury, without any apparent reason, the patient suffered with almost continuous pain in the lower end of the radius. There was no swelling. The recovery from the fracture was apparently normal. In six weeks the man was using his hand in writing. In three months he again began to suffer pain in the lower end of the radius. This local pain continued for three months when swelling was first observed. The swelling was first noticed on the extensor surface of the radius near the joint. Gradually the entire lower end of the radius was expanded. After the swelling had been present about nine months, that is fourteen months ago, an exploratory incision was made into the expanded end of the radius. The surgeon informed the patient that the bone was soft, and that the medullary cavity at the lower end of the radius was occupied by a soft hemorrhagic tissue. The disease was not recognized and the wound was closed without removing any tissue. This is a very important observation, because the exploratory incision into this new growth was made fourteen months before our operation, and as the patient has been observed six years since our operation, we can feel pretty satisfied that internal metastases have not taken place.

Examination.—The lower end of the radius is quite symmetrically expanded. There is no swelling or œdema of the tissues around the bone. The X-ray negative shows a picture corresponding almost exactly to the one reproduced here of the giant cell sarcoma of the upper end of the tibia. The negative demonstrated that the tumor was surrounded by a thin shell of bone intact at every point. The arm was amputated below the elbow.

Pathological Study.—The lower end of the radius is occupied by a very cellular pigmented tumor surrounded by a thin shell of bone; from this shell thin bone partitions extend a few millimeters into the soft tumor tissue. Microscopically the tumor was composed chiefly of giant cells in a matrix of loose vascular connective tissue containing a good many spindle cells. There were many spaces filled with extravasated blood, and there was a great deal of blood pigment, explaining the melanotic appearance of the tumor. The pathological examination demonstrated that the tumor could have been completely removed by resection of the lower end of the radius. It is interesting to note that the few axillary glands removed at the operation because they were enlarged showed blood pigment but no tumor cells.

Result.—March, 1903, five years and nine months after the operation, the patient remains well.

Surg. No. 8409.—Medullary sarcoma lower end of radius. White male aged 28 years. Seven months ago there was a traumatism to the wrist by a baseball. The pain and swelling began directly after this injury and never disappeared. During the last four months the swelling has increased considerably and there has been restriction of motion of the wrist-joint. Palpation and the X-ray negative demonstrated that the tumor has broken through the shell of bone and is infiltrating the surrounding soft parts and tendon-sheaths. For this reason no operation could be considered but amputation, which was done below the elbow. The gross and microscopic pathology of this case correspond to the two cases of medullary sarcoma of the lower end of the radius just described.

Result.—March, 1903, four years and four months after the operation, the patient continues well.

The observations on these seven cases of giant cell sarcoma demonstrate that as yet we have never observed internal

metastases, although in two cases the tumors were recurrent and in one case there was an exploratory incision.

These facts, I think, justify an attempt to remove these tumors by curetting and chiselling, if possible, without destroying the continuity of the bone. If this is not possible the tumor should be dealt with by resection, provided the amount necessary to resect will leave a limb with good function. Amputation is only indicated when the disease has destroyed so much bone, or infiltrated the soft parts to such an extent that resection is either impossible, or if possible, the extent of the necessary operation would leave a practically useless extremity.

These less extensive operations are also justified by cases reported in the literature. These I have discussed in *Progressive Medicine* for December, 1902, p. 150. The cases reported in the literature and those discussed in this communication also demonstrate that if our attempt at removal of the disease is followed by local recurrence the possibilities of internal metastases are apparently not increased, so that as far as observations go, the risk of internal metastases is not increased if in doubtful cases we attempt to cure by curetting or resection. However, it is very important that the surgeon should recognize the pathological variety of the bone tumor. The tumor considered in this paper, the pure giant cell sarcoma, is one in which the less extensive operations are most surely justifiable.

ANGIONEUROTIC ERYTHEMA AND ITS SURGICAL TREATMENT BY NEURECTOMY.

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This condition is apparently one of considerable rarity. The following three cases well illustrate the clinical picture: in one the condition was relieved by division of the nerve over the distribution of which the area of erythema and œdema extended.

Case I.—Angioneurotic œdema of both cheeks, secondary to drainage of the antrum cavities, relieved by neurectomy of the infraorbital nerves. A second area, on the left side of the abdomen relieved by the division of the intercostal nerves. A third area, in the lower abdominal zone, still under observation.

Clinical History.—White, female, aged 17. This patient has been under observation since July, 1900, a period of two years and eight months.

This patient was referred to me by Dr. Randolph, of Baltimore, with the following history: About eight months ago she was under the treatment of a dentist for a cavity in the right bicuspid tooth. Within a week signs of an abscess about the root of this tooth developed. There were pain, swelling and œdema of the gum of the upper jaw, extending into the tissues of the cheek. This swelling persisted for three weeks. At this time an incision was made through the gum over the second molar. The tissues were œdematous, but there was no purulent collection. During the next three months the swelling slightly subsided but did not entirely disappear. The pain and tenderness referred to the right gum and cheek persisted. About this time the second molar tooth was extracted, and a small amount of pus was evacuated. From this opening a slight amount of purulent material continued to discharge. Two weeks later without apparent cause there was considerable swelling and œdema of the cheek between the right eye and the nose. Associated with this there was hemorrhage from the right nares but no discharge of pus. About six weeks ago, without apparent cause, she had a similar attack of swelling and œdema on the left side of the face. For this condition three incisions were made in the gum: one apparently into the antrum, but very little pus, if any, was found. During the last two months she has had peculiar attacks of syncope. These attacks take place sometimes two or three times a day, and there may be a free interval of two to three days or more. The attack seems to consist of apparent loss of consciousness. The patient, however, does not get rigid; she breathes quietly. From the description of the attack one would be inclined to the diagnosis of hysteria rather than of epilepsy. Frequently the attack is preceded by intense pain in the lower left abdominal zone. The patient has lost about twenty pounds in flesh and looks anæmic.

Examination.—The inspection of the face is negative. The second molars on both sides of the upper jaw have been extracted. In the position of the molar there is a small sinus which apparently does not communicate with the antrum cavity. There is at present no swelling or œdema of the gum or cheek. The patient, however, complains of tenderness when the antrum cavities are palpated, and of pretty constant pain referred to both cheeks. Careful inspection of the nasal and pharyngeal cavities demonstrated no pathological condition. There was nothing to indicate any disease of the antrum except the history and the present pain and tenderness. The patient, however, has practically been an invalid during the last eight months on account of this pain and tenderness, and recurrent attacks of swelling and œdema. The abdominal examination was negative, the X-ray shadow showed no stone in the kidney.

Blood Examination.—Red cells, 5,000,000; leucocytes, 12,000; hemoglobin, 85%. Heart and lungs negative. The patient was observed for a week. She was quite hysterical at times, but there were no fainting attacks. She was rest-

less at night and complained constantly of pain and tenderness in both cheeks.

Operation.—July 21, 1900. Ether. The antrum cavities on both sides were opened through an incision above the root of the bicuspid tooth. The size of the opening was about 5 by 8 mm. On opening the left antrum there was very little hemorrhage. The cavity seemed smaller than normal, the mucous membrane was either very œdematous, or it was replaced by granlation tissue. However, as we found no purulent collection in this cavity, I am inclined to think that the pathological condition was one of intense œdema and hyperæmia. On opening the right antrum there was no purulent collection, but the hemorrhage was so profuse from this opening and from the right anterior and posterior nares, that it became necessary to pack the cavity and plug the nares. Every attempt at inspection of the antrum cavity was followed by such profuse hemorrhage that further inspection was abandoned. The patient was allowed to come out of anesthesia, leaving both antrum cavities packed with gauze, and both nares plugged.

The convalescence from the operation was very comfortable. The packing of the antrum cavities and nares was entirely removed on the third day. The patient stated that she was perfectly relieved of the pain and tenderness present before the operation. She left the hospital in two weeks apparently completely relieved. During this time there was no discharge of pus from the antrum sinuses. The patient was advised to keep the small openings into the antra patent.

January, 1901.—The patient returned for observation because of intense pain in the left iliac fossa. These attacks of pain were present only during the period of menstruation. The opening of the left antrum had closed, while the opening into the right antrum was still patent. It is now almost six months since the operation and she has been relieved of the antrum pain and tenderness. After consultation with Dr. W. W. Russell, of the gynecological staff, it was considered best to dilate and curette the uterus. Under ether narcosis the uterus and its appendages were found to be normal. Curetting demonstrated no pathological condition of the lining of the uterine cavity. The operative procedure, however, entirely relieved these attacks of pain up to the present time, February, 1903.

The Development of Angioneurotic Œdema in the Right Cheek.—As the opening into the left antrum had closed and no recurrence of the trouble had developed after some weeks, it was considered justifiable to allow the opening of the right antrum to close. Within a week the patient began to complain of pain in the right side of the cheek; this was associated with almost daily bleeding from the right side of the nose and an area of erythema and œdema appeared in the skin and subcutaneous tissue of the cheek over the right antrum. This area was about the size of a silver dollar, it did not involve the right eyelid nor extend to the nose. There was some fluctuation in the intensity of the redness and the amount of œdema, but the condition never completely disappeared. It had the appearance of an area of skin over an abscess, but the tenderness and pain were not of sufficient degree to suggest a collection of pus in the antrum cavity. There was no fever and no leucocytosis. This area was present on the right cheek only at this time. This area of erythema and œdema which we might call *angioneurotic erythema persistens*, remained in about the same condition from January, 1901, to May, 1902, a period of one year and four months. There were slight changes in color and the amount of œdema, but the area never at any time entirely disappeared. Now and then the pain and tenderness would entirely disappear. In March, 1901, the sinus into the right antrum was reopened. There was no evidence of infection. The mucous membrane was not as œdematous or congested as at the previous operation and there was very little bleeding. This operation relieved the bleeding from the nose and much of the pain and tenderness, and for a short time, the red patch on the right cheek was paler in color.

May 1, 1902.—Although the pain and tenderness in the right cheek were not sufficient to justify an operation, yet when they were added to the marked disfigurement caused by the red and œdematous patch cheek, an attempt at relief seemed justifiable. From the onset of this area of erythema I was inclined to consider the condition similar to angioneurotic œdema, and that without much doubt the original condition in the antrum was not an infection, but a neurosis, i. e. an angioneurotic œdema of the mucous membrane of the antrum cavity. As the infraorbital nerve supplied the area involved, I felt, that its division might relieve the condition, and for this reason on May 1, 1902, the infraorbital nerve on the right side was exposed and divided. Inspection of the tissues through the usual incision beneath the eyelid demonstrated no pathological change. The bony wall of the antrum was exposed, it appeared normal. Almost immediately after the division of the nerve the area of erythema and œdema disappeared. As soon as the patient came out of the narcosis she declared that the pain and tenderness were no longer present. Up to the present time (February, 1903), a period of almost a year, there has been no recurrence of the trouble. Ten days after this neurectomy of the right infraorbital nerve the patient experienced pain in the left cheek and a similar area of erythema and œdema appeared under the left eye. This area corresponded almost exactly to the one just relieved on the right side. The patient was observed for six weeks, and as the condition did not improve, a similar operation was performed on the left infraorbital nerve. The condition of the tissues found on the right side was observed on the left, and fortunately the immediate and permanent relief has been the same.

The Development of Angioneurotic Œdema in the Left Upper Abdominal Zone.—Three days after the neurectomy of the left infraorbital nerve which was performed under chloroform anesthesia a third area appeared suddenly on the left side of the abdomen. It began in the nipple line

at the eighth rib and extended down for a distance of about 6 cm. It corresponded in appearance exactly to the areas described on the cheek. It appeared suddenly one night. The skin over the area was hypersensitive to touch, but slightly anesthetic to pain. The movements of the clothes on this area gave discomfort. Although there was fluctuation in the color, it never disappeared. Observed from June 6 to August 23, the area extended downwards to 1 cm. below the line between the umbilicus and the anterior iliac spine. The width (6 cm.) remained about the same, the length increased from 6 to 19 cm. The patient states that she is always conscious of a sense of discomfort in this area and it is slightly tender to touch. On August 23 under cocaine infiltration an incision was made through the skin and subcutaneous tissue above and to the outer side of the area, dividing everything down to the aponeurosis of the muscle. The erythema immediately disappeared and up to the present time, February 28, a period of six months, there has been no recurrence.

A few weeks after the patient left the hospital, in September, 1902, a similar patch appeared in the left lower abdominal zone, below the one just relieved by neurectomy. This very quickly extended across the middle line and a similar area developed in the right lower abdominal zone. These two areas were similar in their appearance to the others which have been described. Now for the first time the patient began to lose flesh; the blood count, however, showed no marked anæmia: reds, 4,900,000; hemoglobin, 82%. From the first up to the present time careful examination of the heart and other organs showed nothing abnormal. The patient never suffered from tachycardia. The pulse rate, even under narcosis, was always normal. The patient exhibited no other marked hysterical phenomena, except the attacks of syncope which have never reappeared since she came under my observation. On careful questioning I found for the first time that the patient was very unhappy in her home on account of a condition which gave her constant worry and anxiety. For this reason I advised a change of scene. The patient went south and lived in pleasant environment from October, 1902, to January, 1903. During this time she gained in flesh from 107 to 130 pounds. The area of erythema in the right lower abdominal zone disappeared, the area, however, in the left lower abdominal zone has remained about the same, except that it gives less discomfort.

Examination February 27, 1903.—The scars on the cheeks are hardly perceptible. The color of the face is absolutely normal. There is no pain or tenderness referred to either antrum. Sensation in the anesthetic area secondary to the neurectomy of the infraorbital nerves has almost completely returned. The skin corresponding to the first area of erythema on the abdomen is normal in appearance and there are no abnormalities in sensation. The area of erythema in the left abdominal zone as shown in the chart, measures 8.5 by 6 cm.; it is much less sensitive, there is very little œdema; the erythema, however, remains unchanged. The area of erythema in the left abdominal zone has completely disappeared, but the skin over this area is slightly more sensitive to touch than normal. The patient appears in very good health and spirits, and states that the condition does not give her sufficient discomfort to make her wish or be willing to undergo another neurectomy under cocaine anesthesia for its relief.

Remarks.—These areas of erythema differ from the usual cases of angioneurotic œdema first described by Quincke in the persistence of the vasomotor dilatation. Associated with this persistent hyperæmia there is increased sensibility to touch, but decreased sensation to pain; sensation to heat and cold are apparently not disturbed. The areas on the cheek were much more painful and tender than the areas on the abdomen. In all of the areas the erythema could be made to disappear by continuous pressure; the condition, however, reappeared immediately when the pressure was discontinued. The central portion of the area was always of a darker red color, and the redness became lighter towards the periphery. The border of the area was not a sharp line, but there was a zone composed of small patches of erythema and normal skin. The œdema in all of the areas was very slight; in fact, I am inclined to think the swelling could all be explained by the increased amount of blood rather than any actual exudate outside the blood vessels. At the operation I could find no evidence of blood extravasation which is present in forms of purpura hemorrhagica. The condition might be called a localized intense blushing, and most easily explained by vasomotor disturbances; whether peripheral or central it is difficult to ascertain.

This case is remarkable in its multiple areas, in the persistency of the erythema, and is of interest surgically because of the so far permanent relief after neurectomy.

It is to be noted that painting the areas of erythema with collodium decreased the size, but never produced complete disappearance of the area of erythema. For example, on February 27 the area of erythema in the lower abdominal zone measured 8.5 by 6 cm.; on February 28 it measured 6 by 4 cm.

CASE II.—*Angioneurotic Œdema of the Mastoid.*—Female, white, married, aged 42. This patient was apparently at the period of menopause. On April 24, 1902, about ten months ago, she consulted me because of pain and tenderness over both mastoids, and a slight area of redness and œdema which comes and goes over the right mastoid. The pain over the right mastoid extends down the neck to the right shoulder and is associated with headache. The pain is fairly constant and at irregular intervals becomes quite intense. It rarely keeps the patient awake at night. There is no history of any previous trouble with either ear.

Examination.—Both mastoids are slightly tender, the right more so. The tissues over the right mastoid are perhaps slightly œdematous and beneath the skin the lym-

phatic gland is felt to be slightly enlarged. The redness which, the patient states, was present yesterday is absent to-day. There is no redness or œdema over the left mastoid. The patient was seen at intervals for ten days. On two occasions, over the right mastoid there was an area of skin of the size of a fifty-cent piece intensely red in color and slightly œdematous. Associated with this the tenderness over the mastoid was slightly increased. The erythema and œdema were out of proportion to the local tenderness. There was no fever or leucocytosis. Dr. Randolph could find no pathological condition in the nares or pharynx. The Eustachian tubes were patent. He removed from the external auditory canal a large plug of cerumen, this plug pressed upon the apparently normal tympanic membrane; it was very hard, and after its removal he observed a slight erosion of the epidermis of the canal. This relieved the condition only for a few days. Clinically the area of erythema and tenderness over the mastoid resembled the one described in the previous case over the antrum, except the redness now and then completely disappeared. The erythema did not extend to the auricle. The urine contained a slight amount of albumen and a few hyaline casts.

Operation.—April 24, 1902. Under cocaine infiltration an incision was made over the mastoid. It is to be noted that on the day of the operation the erythema and œdema disappeared. At the incision all the tissues were found to be normal, except a slightly enlarged and hyperæmic lymphatic gland and the veins which came through the small foramen of the mastoid seemed to me to be slightly larger than usual. I could make out no pathological change in the periosteum or outer table of the mastoid, and for this reason did not chisel the bone. The gland mentioned was removed and the veins ligated.

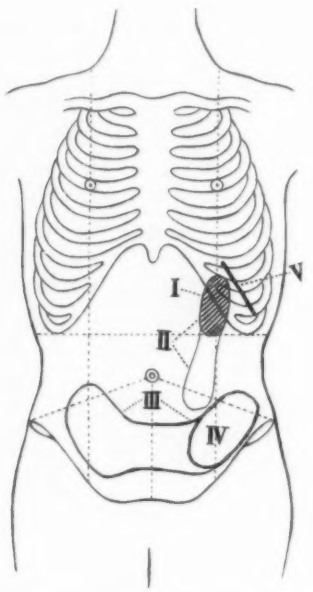
This patient has been observed up to the present time, March 1, 1903. After the operation the patient was free from pain and erythema for about three weeks, since then there have been intermittent attacks. The tenderness is apparently getting less; the erythema appears at longer intervals and is less intense. The pain seldom is referred to the neck and shoulder; the headaches are less. Now and then the pain extends to the ear and over the parotid area. The condition on the left side is present at rare intervals, and is much less severe. Now and then the attacks are associated with slight flushing of the face and ears. The patient informs me that she rests well at night. The attacks of pain and erythema usually come on in the afternoon and evening, especially when she has had a busy day and especially if she has suffered any unusual anxiety. Now that the patient understands that the condition is not a serious one she states that it does not give her sufficient discomfort to make her desire any operative interference. Painting the area with collodium has little or no effect. Covering the area with antiphlogistine has no effect.

The object of my operation in this case was to exclude any inflammatory condition of the mastoid cells; as it is difficult to chisel into the mastoid without a general anesthetic, I did not consider that we were justified in view of her chronic nephritis to give ether for this procedure. The signs could all easily be explained by a local vasomotor dilatation.

CASE III.—*Angioneurotic Œdema of the Elbow Joint* (Idiopathic hydrops articulorum intermittens).—I have reported this case in Progressive Medicine, December, 1900, p. 201, with the discussion of the literature on intermittent hydrops. It is my only observation. The patient was referred to me by my colleague, Dr. Smith, of Baltimore. She was a woman 27 years of age; married; no children. Following slight trauma, two years ago, over the anterior surface of the left tibia two ulcers formed, and healed slowly. A year ago she had a slight trauma to the left elbow. Following the trauma it was slightly swollen, since which time she has had remittent attacks of swelling of this elbow-joint. The periods have been pretty regular. The swelling is sometimes associated with slight pain and restriction of motion and the feeling of tingling and numbness in the fingers. On one occasion during a recent attack, without trauma, the other elbow was similarly affected. I saw her at the end of an attack, and there was an area of œdema, confined more to the subcutaneous tissues than to the skin, over the external condyle of the humerus. There was little effusion in the elbow-joint. The skin was normal. Fearing that perhaps there might be a beginning new growth of some kind, although from the history and the careful examination I felt this could almost be excluded, I advised an exploratory operation. A few days before the operation most of the œdema disappeared. At the operation all the tissues were normal except the periosteum. In stripping this from the bone the outer table of the humerus seemed a little rougher than usual. Chiselling into the external condyle, the bone was normal. The wound healed kindly. Following the operation the œdema disappeared in a few days. About three weeks later, without apparent cause, she suddenly had the same swelling of the elbow, associated with a similar one, but to a slighter degree, of the opposite elbow, and a definite area of œdema of the dorsum of the hand between the thumb and index finger. It disappeared after four days. Here we have, therefore, a history of a localized acute œdema, most marked in the left elbow-joint, involving the periarticular tissues. associated with slight effusion, recurring at irregular intervals, and on two occasions associated with a similar condition of the opposite elbow and skin of the dorsum of the hand. The patient was otherwise healthy, but of a marked nervous temperament and very hysterical. In this case the operation and the histological examination of the tissues demonstrated no pathological change. Previous prolonged treatment with mercury and iodide practically exclude lues. I must confess

that until I read Schlesinger's[1] article I was at a loss to explain the condition.

Note April, 1903.—The operation was performed in May, 1900. The subsequent slight attack in June is noted in above history. In July the patient observed that she did not menstruate at the usual time. I saw the patient in October: there were signs of pregnancy in the third month and the patient was absolutely free from all joint symptoms. She was delivered later of a healthy child. In April, 1903, almost three years after operation, this patient again came



AREAS OF ANGIONEUROTIC ŒDEMA OBSERVED IN CASE I.

I. = Area observed May 15, 1902.
II. = Extension of Area I to August 23, 1902.
V. = Incision made August 23, 1902.
III. = Area September 20, 1902.
IV. = Present area, February, 1903.

under observation. An examination demonstrated an area of tuberculosis of the skin of the right forearm of six months' duration, and an abscess over the right sternoclavicular joint of three months' duration. These conditions have been relieved by operation, and the pathological study has proven their tubercular etiology. The left elbow during all this time has never given her any trouble, and she has observed no recurrence of the symptoms mentioned in the former history. Although intermittent joint hydrops is now and then observed as an early symptom of joint tuberculosis, and although the present lesions are undoubtedly tubercular, I do not think that these facts prove that the original lesions were symptomatic of a tubercular focus, but can be more easily explained as due to an angioneurotic disturbance.

Note April 12, 1903.[2]—On Monday evening, March 2, the patient was exhibited before the Medical Society. On that evening there were no areas of erythema, except the one marked "iv" on the abdomen. The following Tuesday night the patient's attention was called to the left arm between the shoulder and elbow by a sensation of pain. The following morning she observed the typical red area which rapidly extended between the shoulder and elbow on the outer side of the arm. There was sufficient pain in this area to keep the patient awake Wednesday night. Thursday morning the patient painted the area with collodium which relieved the discomfort.

Examination Thursday Afternoon.—The area has been observed 36 hours. It occupies the middle of the outer surface of the arm, it is oblong in shape, 11 cm. long and 6 cm. wide, it is covered with collodium. There is unquestionably some œdema which extends a little beyond the area of erythema. This arm, the left, measures 5 mm. greater in circumference. The spot in the left lower abdominal zone (see Fig. iv) has faded since the use of collodium.

On March 10, five days later, the patient was taken with quite intense pain in the abdomen which was soon followed by the appearance of the usual menstrual flow. In a few hours she had the usual prodromal sensation of pain and prickling in an area on the left forearm. The patient being now familiar with the fact that this sensation would soon be followed by a patch of erythema painted the spot immediately with collodium. This relieved the pain very much, and when I examined the area 24 hours later, it was but slightly erythematous.

Note April 1, 1903.—The area on the arm has changed very little, the area on the forearm has extended to the wrist. The condition has not recurred in any of the previous spots.

[1] Mittheilungen aus den Grenzgebieten der Med. und Chir., 1899, i, p. 441, reviewed in Progressive Medicine, December 1900, p., 195.

[2] Reported before the Johns Hopkins Medical Society March 2, 1903, with exhibition of case 1.

THE JOHNS HOPKINS HOSPITAL BULLETIN.

The Hospital Bulletin contains details of hospital and dispensary practice, abstracts of papers read, and other proceedings of the Medical Society of the Hospital, reports of lectures, and other matters of general interest in connection with the work of the Hospital. It is issued monthly.

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SUMMARIES OR TITLES OF PAPERS BY MEMBERS OF THE HOSPITAL OR MEDICAL SCHOOL APPEARING ELSEWHERE THAN IN THE BULLETIN.

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HUGH H. YOUNG, M. D. Treatment of Calculus of the Lower End of the Ureter in the Male.—*American Medicine*, August 9, 1902.

NOTES ON NEW BOOKS.

Book on The Physician Himself and Things that Concern his Reputation and Success. By D. W. CATHELL, M. D. The Twentieth Century Edition. Being the eleventh edition revised and enlarged by the author and his son, WILLIAM T. CATHELL, A. M., M. D., Baltimore, Md. (*Philadelphia: F. A. Davis Company, Publishers, 1902.*)

The value of this book to those who wish information upon the practical side of medicine is well attested by the fact that ten previous editions have been issued. The present edition has been carefully revised and is very suggestive and readable. While one might possibly prefer that the number of poetical and proverbial quotations had been fewer or less conspicuously printed, this is simply a matter of taste. This abundance of quotation tends to render the meaning more emphatic and to stimulate the attention of the reader by the introduction of a pithy remark or an illuminating sentence.

The International Text-Book of Surgery. In two volumes. By American and British authors. Edited by J. COLLINS WARREN, M. D., LL. D., F. R. C. S. (HON.), Professor of Surgery, Harvard Medical School; and A. PEARCE GOULD, M. S., F. R. C. S., of London, England. Second edition, thoroughly revised and enlarged. Vol. I. General and operative Surgery. Royal octavo of 965 pages, with 461 illustrations, and 9 full-paged colored lithographic plates. Vol. II. Special or regional surgery. Royal octavo of 1122 pages, with 499 illustrations, and 8 full-paged colored lithographic plates. (*Philadelphia and London: W. B. Saunders & Co., 1902.*)

It is hardly necessary to say that this text-book, containing as it does, articles by several of the most eminent surgeons of England and the United States, represents much of what is really the best modern surgery. Those sections which have been written by men who have made the special branch treated, their life-

work, are naturally the ones most worth consulting. The article by Drs. Bull and Coley, for example, presents hernia as thoroughly as could be desired, and without giving up many pages to the consideration of trusses, a mistake too often seen in some of the more recent monographs on this subject. Further, in cases where the operation, originally described by any surgeon has been, in the course of time, materially modified, due note of the fact has been made. The simplification of Dr. Halsted's original operation for hernia is thus duly noted. Appendicitis, as might be expected, is treated by Dr. McBurney, and the chapter is one of the best in the book. The advances made by American surgeons are nowhere better illustrated than in the treatment of this affection. Two of the sections by English surgeons deserve special attention. Few portions of the book are so readable as Mr. Pearce Gould's section on the Surgery of the Neck, particularly because it lays so much stress on the embryological factors concerned in this region. His descriptions are clear, and his operative measures thorough. Mr. Bland Sutton, in forty pages, gives a remarkably concise account of Tumors, although Carcinoma can hardly be said to receive adequate attention, even in general terms, with less than five pages devoted to it.

Neither the article on Prostatic, nor that on Ureteral Surgery, contain an adequate account of the recent advances in those branches. Bottini's operation is barely mentioned, nothing being said as to the results obtained with it, or of the methods of procedure. In the section on the Surgery of the Nose, no mention is made of the methods of Paraffin injection for the restoration of deformity.

The chapter on diseases of the Lymphatic System is a disappointment. The statement at the very outset, for example, that "the peritoneum, the pleuræ, and the joints . . . are in direct communication with the lymphatic vessels, and may be regarded as reservoirs of the lymph," causes no little surprise in view of the recent pretty conclusive demonstration that the lymphatic vessels, like the blood-vessels, form a completely closed system, and that there is no such direct communication between them and the peritoneum, for example, as has been hitherto believed to exist. Although mention is made of the danger of wounding the thoracic duct in neck operations, we are not told just where to look for that vessel. The possible lesions of the spleen are enumerated, but their symptoms are scarcely referred to, and no mention is made of differential diagnosis.

In spite of the minor defects mentioned, this text-book probably covers the necessary ground as well as any available in English, of the same size.

Atlas and Epitome of Diseases of the Mouth, Pharynx and Nose. By DR. L. GRÜNWALD, of Munich. Edited by JAMES E. NEWCOMB. (*Philadelphia: W. B. Saunders & Co., 1903.*)

Grünwald's treatise deserves a prominent place among the many works now appearing on the nose and throat. It represents the result of many years of careful observation and contains the results of much individual work. The work is divided into two parts; the first consisting of a series of colored plates, the second a brief but comprehensive review of the diseases of the nose and throat.

The plates are well selected and comprise the diseases commonly seen. The description of the lesions, together with the short history of each case, makes them quite clear. The drawings also are very well reproduced.

The second part of the work is to be especially commended. Much of the work is original with the author, such as the chapter on focal diseases of the nose which seems an epitome of his book on "Nasal Suppurations." The chapter on mouth breathing is interestingly written. The relation of mouth breathing to certain so-called neuroses as pavor nocturnus, nocturnal incontinence and some cases of epilepsy is referred to. The inflammations and hyperplasias of the pharyngeal lymphatic ring receive good treatment in the appendix. The author emphasizes the importance of these structures as a "*locus minoris resistentiæ*" for the entrance of pathogenic microorganisms, which cause widespread inflammations in the body, as in the serous membranes, the joints and bones.

A Text-Book of Anatomy by American Authors. Edited by F. H. GERRISH, M. D., Professor of Anatomy in the Medical School of Maine Bowdoin College. Second Edition. (*Philadelphia and New York: Lea Brothers & Co., 1902.*)

This text aims to fill a place "between the extremes represented on the one hand by pocket manuals with their flavorless condensation, and on the other hand by encyclopedias of universal inclusiveness" (Gerrish, p. 5).

In other words the authors modestly place before us an "elementary work on anatomy" (Gerrish, p. 57). and as such the work is a success. The text is well and clearly written. The illustrations, which are good, are largely from the French anatomy, Testut. The relation of muscles to skeletal attachments illustrated diagrammatically is one of the most valuable and suggestive features of the work, and should prove a great aid to the student in making free-hand drawings on outline charts, such as are being used in our leading medical schools in America. In Germany such drawings have constituted a valuable part of the student's work for some years.

Many of the diagrams are excellent. The engraving of names of parts directly upon them is very commendable. The areas of muscular attachments to the bones, illustrated in the chapter on "Osteology" is a good feature of the work.

Since five of the six authors are or have been surgeons, it is not surprising that unusual attention has been given to surface, relational, and surgical anatomy.

In 1895, in Basel, an anatomical nomenclature was adopted by a body of the best anatomists of the world. These terms, the Basel Nomina Anatomica, [B NA] seem destined rapidly to become international. They have been employed in revising old German texts. The new atlases of Spalteholz and Toldt have incorporated them, and their introduction into our leading American colleges, together with the translation of Spalteholz, is rapidly bringing these terms into use in America. The adoption of this nomenclature in the part on the "Cerebro-Spinal Axis" would have rendered this clearly written chapter to a much greater degree a preparation for more extensive works and won for the book a much more favorable recognition in Europe.

The authors have not aimed at completeness nor at great detail in description, but with the possible exception just mentioned, they have accomplished well their avowed purpose, that of presenting an elementary text-book of anatomy.

B. D. M.

Golden Rules of Refraction. By ERNEST E. MADDOX, M. D., F. R. C. S. Ed. Golden Rule Series No. XII. (*Bristol: John Wright & Co.*)

This book, like its predecessors, contains much information in a small compass. It is intended to give general practitioners who are commencing the study of refraction, a general conception of the work before them. We are gratified to see that the author recommends bifocal glasses to all persons who might otherwise be compelled to use two sets of spectacles. We are also gratified to learn that they were invented by Benjamin Franklin and popularized by Sir Joshua Reynolds.

The Story of a Living Temple. A Study of the Human Body. By FREDERICK M. ROSSITER, B. S., M. D., and MARY HENRY ROSSITER, A. M. (*Chicago, New York, Toronto: Fleming H. Revell Company, 1902.*)

This attractively printed and thoroughly well-intentioned volume is a distressing instance of an overworked metaphor. Starting out with the scripure text, "Know ye not that ye are the temple of God, and that the spirit of God dwelleth in you? If any man defile the temple of God, him shall God destroy; for the temple of God is holy, which temple ye are," thirty-eight chapters follow devoted wholly to an elaboration of the different phases of this simile. The titles of the chapters are often highly imaginative, such as "The Wonders of the Temple," "How the Wall of the Temple Can be a Living Wall," "The Breathing Rooms" (the lungs), "Winking Valves and Living Pumps (the alimentary canal), etc. The book displays much careful work and is imbued with a strong religious tone. It is popular rather than scientific.

A Treatise on Diseases of the Anus, Rectum, and Pelvic Colon. By JAMES P. TUTTLE, A. M., M. D., Professor of Rectal Surgery in the New York Polyclinic Medical School and Hospital; Visiting Surgeon to the Almshouse and Workhouse Hospitals. (*New York: D. Appleton & Co., 1902.*)

From the point of view of extensive personal observations, of the record of others' work, of arrangement, and of illustration, Dr. Tuttle's book practically attains to the modern ideal of a perfect text-book.

In an introductory chapter, the anatomy of the rectum is well described, the cuts illustrating the anatomical divisions being particularly clear.

Of the many excellent sections in the book, special reference may be made to those on Tuberculosis, on Fistula and on Hæmorrhoids. There is an interesting note on Hyperplastic Tuberculous Colitis, that peculiar form of the disease which has recently been brought to general notice, especially by the French writers. The chapters on Fistula, together with that on Perirectal Abscess, are perhaps the best in the book. The illustrations in this section are unusually good, showing most clearly the varieties of fistulous tracts, and the essentials for treatment. It is interesting to note that 50 per cent of the fistulas seen by Dr. Tuttle were in persons, either at the time, or later, affected with tuberculosis.

The thorough chapter on Hæmorrhoids ends with Dr. Tuttle's conclusion that "on account of its applicability, the ease and celerity with which it can be applied, and its uniformly good results, the clamp and cautery easily stands first among the operations for hæmorrhoids."

The only illustrations in the book that are unsatisfactory, are the microscopical drawings of carcinoma and sarcoma of the rectum.

The brief section on Rectal Feeding, which includes a number of appropriate formulæ, ought to be very useful.

Throughout the book, operative measures are very fully described, all the leading methods being given in detail, and followed by a careful weighing of the facts for or against a given procedure. This is well illustrated in the section on operations for excision of the rectum, where the steps in the evolution of the operation now generally done, are shown by a series of cuts.

No one interested in this branch of surgery can afford to be without this book.

A Manual of Medical Treatment or Clinical Therapeutics. By I. BURNEY YEO, M. D., F. R. C. P. Two volumes. 10th edition. (*Chicago: W. T. Keener & Co., 1902.*)

To the student who is taught throughout his course that diagnosis is the chief end of the medical man, and who has, nevertheless, an instinctive feeling that when he is actually in practice, he will be expected to treat sick people, Dr. Yeo's book is a great blessing. It is probably the most acceptable work of its kind, largely because treatment is approached from the point of view of the disease and not from that of the drug. The care of the successive stages of a disease is described in detail. This is well illustrated in the section on dysentery and on bronchitis, where each indication and its appropriate treatment are fully outlined.

Dr. Yeo is evidently a polypharmacist, providing a remedy for every symptom, and often recommending heavy formulæ, a tendency from which American physicians are slowly getting away. As is so customary in England, the dose of nearly every mixture is a tablespoonful.

Sixteen pages are devoted to the antiseptic treatment of tuberculosis. Aside from the prevalent disbelief in this, the very fact that good observers get such widely divergent results with the same drug, proves the inefficacy of most of them. One is surprised to note the emphasis on the use of styptics in hæmoptysis, when it is borne in mind that the researches of Bradford, Dean and Andrews have shown that these, by raising the pressure in the pulmonary circulation, may actually increase the tendency to hæmorrhage.

In the treatment of typhoid fever and of appendicitis, the American physician will probably disagree with Dr. Yeo. The antiseptic treatment of typhoid, now so generally acknowledged to be unavailing, is still warmly advocated by Dr. Yeo. Although he expresses surprise that any of his favorite "Chlorine Mixture" should reach the intestine, he still advocates its use on empirical grounds, and even suggests that some of it is absorbed into the blood. Further, without a word as to the tonic effect of the cold bath treatment, three pages are given to the criticism of this method as a routine, although acknowledgment is made that the author has had no opportunity to test the value of the statistics adduced in its favor. No mention is made of calcium chloride in the hæmorrhage of typhoid, a heavy gallic acid mixture being suggested instead.

Dr. Yeo shares the general English conservatism as to the treatment of appendicitis, believing that it is regularly possible to recognize the cases which require operative measures early. He recommends opium in the acute suppurative form in spite of the repeated plea from the surgeons against it on account of its masking the symptoms. Aperients are advocated for "mild" attacks.

Dr. Yeo's book may be recommended to every one who wishes to treat disease intelligently and thoroughly. It is well arranged and well indexed, and due credit is given to every author whose ideas are quoted.

Biographic Clinics. The Origin of the Ill-health of De Quincey, Carlyle, Darwin, Huxley and Browning. By GEORGE M. GOULD, M. D., Editor of American Medicine. (*Philadelphia: P. Blakiston's Son & Co., 1012 Walnut St., 1903.*)

The thesis of this attractive volume is that the five men whose names are mentioned upon the title page suffered from eye-strain and consequent neurasthenic symptoms. As no mention was ever made of the presence of this condition by their biographers or any record of an examination into the actual refractive defect by a competent oculist exists, it is of course necessary that the author should gather his information from such biographic details as have been given in their published letters. From the abundant evidence which he obtains as to their sufferings whenever they were compelled to use their eyes continuously he infers with every shadow of reason that they all suffered from eye-strain in consequence of their severe studies or excessive literary labors. If, in addition, an error of refraction had been demonstrated in each individual case considered, the strength of the argument would have been increased. Enough, however, seems to have

been gathered to render the truth of the contention extremely probable even if actual proof is not presented.

The true value of the book, however, lies in the concluding chapters on "Biliousness and Headache," "Some Neglected Points in the Physiology of Vision," and "Astigmatism and Eye-Strain." These we commend to every neurologist for careful and thoughtful study. The author writes earnestly and forcibly, as he always does, and his facts and arguments are most weighty.

Diseases of the Pancreas and their Surgical Treatment. By A. W. MAYO ROBSON, F. R. C. S., Senior Surgeon, Leeds General Infirmary, and B. G. MOYNIHAN, M. S. (Lond.), F. R. C. S., Assistant Surgeon, Leeds General Infirmary. (*Philadelphia and London: W. B. Saunders and Company, 1903.*)

The interest at present accorded to disease of the pancreas is shown by the frequency with which this subject has been recently assigned for the discussion of various medical associations. The coming Congress of American Physicians and Surgeons will give an entire session to the subject. Further evidence of this interest is furnished by the work of Mayo Robson and Moynihan devoted particularly to the surgical aspect of diseases of the pancreas. The book, which is printed in attractive form, is dedicated to the surgeons of America.

A chapter is given to the anatomy of the gland and is followed by a review of the experimental and in part clinical studies undertaken to establish the relation of the organ to carbohydrate metabolism. Throughout the work the authors have made use of the abundant clinical material which has come under their own observation, and at the same time cite numerous cases from the abundant literature of pancreatic disease. The relation of chronic pancreatitis to cholelithiasis has received especial attention. Mayo Robson has in previous publications emphasized the frequency with which gall stones in the common duct are associated with such induration of the pancreas that the diagnosis of malignant disease is made at operation. In twenty-four cases in which this condition was recognized all save one recovered. It seems that the writers give undue importance to the symptomatology and treatment of the associated chronic interstitial pancreatitis, which in great part at least are those of the primary cholelithiasis.

It is unfortunate that the term subacute has been introduced into the classification of inflammations affecting the gland. The classical terms, suppurative pancreatitis, hæmorrhagic pancreatitis, and gangrenous pancreatitis, a late stage of the hæmorrhagic lesion, represent well defined pathological changes. The term subacute has been used to designate those cases which do not terminate fatally within a few days and is not applicable to any readily definable lesion.

The book in large measure fulfils the purpose of the writers to review the work of the past and to point out the need of further advance.

Atlas and Epitome of Human Histology and Microscopic Anatomy. By PRIVATDOCENT DR. J. SOBOTTA, of Wurzburg. Edited, with additions, by G. CARL HUBER, M. D., Junior Professor of Anatomy and Histology, and Director of the Histological Laboratory, University of Michigan, Ann Arbor. With 214 colored figures on 80 plates, 68 text-illustrations, and 248 pages of text. Cloth, $4.50 net. (*Philadelphia and London: W. B. Saunders & Co., 1903.*)

The illustrations in this work represent, with few exceptions, sections of various organs of the human body. Unusual care was used in making the drawings. The preparations were photographed, from the photographs outline drawings were traced on tracing-paper and then transferred to drawing paper, and then the drawings were finished from a direct study of the sections. In this way various details were accurately portrayed. Nearly all the illustrations in the volume were made under relatively low magnifications, such as are used by the majority of students in the general microscopic courses. Since most of the preparations were made from human tissues obtained from individuals who had been executed, and the illustrations, both the colored and the black and white, are clear and accurate, the atlas offers a ready means of getting a good idea of the appearance of normal human tissues, hardened, sectioned and stained. It is therefore likely to be of especial advantage to those who find it difficult to get first-rate specimens of normal human tissues.

As an aid to the student in getting a good conception of the finer structure of the body, the atlas will be found lacking in several important particulars. Very few illustrations of teased preparations are given. Diagrams are infrequent, although several excellent ones of the salivary glands and pancreas show their value. No attempt is made to illustrate by means of reconstructions or dissections the third dimension. It is essentially a two-dimension histology.

The text is clear, concise and as accurate as one could well find where brevity enforces dogmatism. No references to the literature are given. The English of the translation is smooth and pleasant. The additions which the editor of the translation has made are of such value that one wishes he had used his hand more freely.

C. R. B.

How to Succeed in the Practice of Medicine. By JOSEPH MCDOWELL MATHEWS, M. D., LL. D. (*Louisville: John P. Morton Company, 1902.*)

This book has an eye single to business and aims to inform the young physician as to the personal side of the practice of medicine, that is, how he should conduct himself so as to attain professional success. This success is not of a large sort; it is pecuniary success rather than scientific achievement. The volume is marred by many rather pointless tales and anecdotes and "to relieve the monotony," we are told by the author, "a jocular vein is interspersed here and there." It is also rendered extremely tedious by attempts at fine writing. The words "home" and "wife" always seem to set the author off and he goes on indefinitely after this fashion: "A beautiful woman—the Creator's most perfect handiwork! The attempt of the artist to catch the rich coloring of her cheeks, the depth of her great blue eyes, the radiance of expression, the turn of lip, the taper of fingers and the luster of her hair is futile, and his brush falls to the ground in utter ignominy. To paraphrase: 'God possibly could have made a more beautiful thing than a beautiful woman but he never did.' Think of her power. A man will go on bended knee to her; the clasp of her hand can drive him mad, and to touch her lips he would sacrifice wealth and honor. Madness, despair, drunkenness and sorrow go in her wake, for men would creep in mire and filth to do her command. The history of the world recites more than one Anthony and Cleopatra, and thousands have gone to their death by drink, by opiate and by the pistol shot as the result of her presence and influence. No, not alone for beauty of face, but rather beauty of character, should you select the one who is to share your burdens as well as your joys. One possessed of natural grace, of comely appearance, of health; whose disposition is as charming as her traits; whose laughter is contagious. One whom men speak well of and women praise. Educated, refined and of quick perception, not given to gossip or an evil tongue; one who believes that 'this is a beautiful world and filled with pleasant people,' one who knows not cant, hypocrisy or deceit and whose manner is that of the perfect lady. Take her, for she will be an honor to you and her glory will be everlasting."

The advice given in the book is sensible, and the writer shows much familiarity with practical matters. It is attractively printed

and would be readable if it were written in a less sentimental vein.

Physiology at Harvard. By WILLIAM TOWNSEND PORTER, M. D., Associate Professor Physiology, Harvard Medical School. Second edition. (*Cambridge, Mass.: The University Press, 1903.*)

The object of the work is clearly and concisely stated in the preface, which is as follows:

"This book is written to explain a new method of teaching, sound in theory and feasible in practice; to provide the Harvard Medical School with a precise account of the work done by each student in Physiology; and to create for students and instructors alike a working-plan by which they may find their way unvexed through much detail."

The "new method" is designed to make of the student an observer rather than a listener, to teach him to seek evidence not authority, to study nature not books.

The means for achieving these results, in other words, the "new method" itself consists essentially of the following factors:

1. *Laboratory Work.*—This forms the most important part of the course and is supplemented by frequent "conferences" (or informal recitations) and written tests. The number of experiments performed by the student is very considerable and at the end of the course he is required to pass a practical examination. An interesting feature is the substitution for the usual "laboratory guide" of *printed accounts of the experiments as originally performed.*

2. *Demonstrations.*—These are given weekly to small groups of students.

3. *Recitations, &c.*—The student's work is correlated and systematized by means of conferences and recitations which are didactic in character, being "in no sense examinations"; such lectures as are given are merely explanatory of the laboratory work.

4. *Theses.*—Each student is required to write a thesis on a topic assigned to him and for which he is provided with a bibliography. The best of these theses are read and discussed in class.

The educational value of the various factors which make up the "new method" cannot be questioned, and that they are well adapted to the end in view is obvious. But why the term new is employed is not altogether clear. Neither the object nor the factors impress one as being altogether novel. If it be the proportions in which the factors are mixed then this method is in a sense "new." But would it not be more appropriate to speak of it as the "Harvard method"?

Dr. Porter implies that the spirit of research and the desire to acquire knowledge first hand is a distinctive feature of the work at Harvard. When, however, such a spirit pervades the student body may it not be attributed to the personal influence of the teaching staff rather than to any educational agency such as the method in question?

Certain features of the Harvard method call for special comment.

1. It seems possible that there may be cases in which the lecture may have some advantage over what we may call the didactic recitation. It should not be forgotten that there are lecturers who are not automatic text-books, but living men full of a contagious zeal for science and experimentation. Again, there are perhaps as many teachers who cannot conduct a recitation properly as there are poor lecturers. Lastly, it may be urged that the value of the recitation varies inversely as the size of the class if not with the square of the size.

2. The advantage of preparing a thesis to the individual who writes it is certainly great, but may not the value to the class of listening to the reading and discussion of such theses be questioned?

3. A large amount of laboratory work is of course very desirable, and those institutions in which such work is not already a special feature would do well to follow Harvard along a path made comparatively inexpensive by Dr. Porter's persistent efforts to lessen the cost of class apparatus.

To those engaged in the teaching of physiology a word from one of our foremost medical schools is always welcome, and it might be equally helpful if one or more of the other great laboratories of this country would provide us with an equally complete account of the ways in which they are solving the ever present problem of instruction in physiology. D.

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For particulars, apply by letter to Dr. E. N. Brush, Physician-in-Chief and Superintendent, Sheppard and Enoch Pratt Hospital, Station "A," Baltimore.

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